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RUPTURED AORTIC ANEURYSMS IN CHILDHOOD, WITH THE REPORT OF A CASE.

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ANEURYSMS are so infrequent in children that it is advisable to place on record each example met with. Such a record is especially desirable when detailed pathological reports are available.

CASE HISTORY.

Alfred G—, aged 4½ years, was admitted to the Paddington Green Children's Hospital, October the 6th, 1915, complaining of pain in the chest.

Family history.—The father is a soldier, and is healthy. The mother is a sensitive, nervous woman, but in good general health. She has had only the one pregnancy. No specific history in either parent is elicited.

Previous history.—He was a full-time infant, breast-fed for two months, then brought up upon barley-water and milk. He spoke, and cut his teeth early, but he did not walk until three years of age. He has never snuffled, and has had no skin troubles. The unusual shape of his head and ears and the looseness of his joints attracted attention early in infancy. Also, since early infancy there has been a peculiar gurgling in his abdomen, accentuated by lying on his back. His appetite has never been good, and when he has eaten much he has been troubled by pain in the chest and attacks of vomiting. He has been subject also to periods of constipation

alternating with diarrhœa. Shortness of breath showed itself as soon as he started to walk, and a thumping action of the heart was noticed after a fright a few weeks before admission to the hospital. At two years of age he was operated upon for inguinal hernia. With the exception of chickenpox, he has had no acute illnesses. Since eleven months of age he has been troubled by an irritative cough, which becomes worse when he lies down. Occasionally he has had short attacks of pain, with panting breathing. Because of shortness of breath he has never been able to run, nor to walk over half-a-mile at a time. Owing to the cough he has always been under a doctor's care, but no mention of heart trouble was made to the mother previous to admission to the hospital.

Condition on admission.—He is a somewhat under-nourished child, walking into hospital, and not acutely ill. Mentally he is old in many ways. He is sensitive and rather silent, but has a quaint method of expressing himself, and a sense of humour of his own. He has no idea of how to play with other children, due possibly to the fact of his being an only child.

Head.—The expression of his face and the shape of his head attract attention at once. The forehead is full and high, but not bossed. There is an increase in the biparietal diameter, so that the ears are turned outward from the head. The occipital prominences are also conspicuous. The ears are large, and lack some of the normal folds of the pinna. The bridge of the nose is somewhat depressed. The hair of the eyebrows is scanty. The epicanthic folds are in excess, and there is a slight Mongolian slant about the eyes. The palate is high arched. The mouth is open, and the facies generally suggests adenoid obstruction.

Extremities.—The joints are lax and flail-like with definite subluxation of the elbows. The fingers are slightly clubbed.

Spine.—Lordosis is present on standing.

Chest.—Pigeon-breast deformity and a marked Harrison's groove are present. The right side of the chest is fuller than the left, yet expansion is the better on the left.

Abdomen.—The abdominal wall is flaccid. The spleen is palpable at the costal margin. The liver is not enlarged.

Lungs.—The percussion note is slightly impaired at the left base and the air entry is deficient, but the latter improves when the child is turned on the right side. The breath-sounds are exaggerated over the entire right side.

Heart.—There is no cyanosis and no dyspnœa at rest in bed. Pulsation is visible in the epigastrium and over the rib cartilages to

the left of the epigastrium. The apex-beat is in the sixth intercostal space ½ in. external to the nipple line. Over the inner portion of the second, third and fourth right intercostal spaces there is visible and palpable pulsation and a corresponding dulness, which extends about 2 in. from the right border of the sternum. On the other hand, cardiac dulness is not marked to the left of the sternum, and pulsation is not so evident. The area of pulsation measures 6 in. transversely. No thrill is felt on either side. At the apex both sounds are heard and there is no murmur. In the third intercostal space on the left a soft systolic blow is heard, while over the dull area to the right of the sternum the murmur is loud and the second sound is exaggerated. The systolic bruit is especially noticeable in the region of the right nipple, but it is not conducted into the right axilla. The pulse is small and regular.

Tracings of the radial pulse with the Mackenzie polygraph are normal. When the receiver is placed over the cardiac apex and over the right nipple the tracings are similar, suggesting expansile pulsation on the right side, and not merely a communicated impulse, as from a new growth.

Urine examination is negative.

Wassermann reaction is negative.

Fluoroscopic and radiographic examinations.—A pulsating mass is seen to the right of the sternum, corresponding to the dull area on percussion. The plate indicates also a large mass, which extends out into the position of the right lung, with convex surface toward the right.

Progress.—The child was in the hospital five weeks, during the first three of which he had an afternoon temperature ranging between 100° and 102° F. The cause of the pyrexia remained unexplained. He had occasionally slight catarrhal signs in the lungs. A hacking cough was present continually, and at night this became paroxysmal, like whooping-cough. The pulse ranged between 76 and 110, the higher rate occurring only when the temperature was elevated. Similarly, the respirations were between 20 and 36. He was up about the ward after the temperature had settled, and he showed no symptoms of distress.

A second fluoroscopic examination was made before he was discharged, with the following report: "The cardiac apex is in the normal position. On the right side there is an expansile mass which moves synchronously with the apex-beat. On the left side of the chest a pale shadow extends to a level well above the apex of the heart. The diaphragm moves well on both sides."

After discharge from the ward he was under observation in the out-patient department until his death. He had attacks of diarrhœa alternating with constipation, and occasional sudden

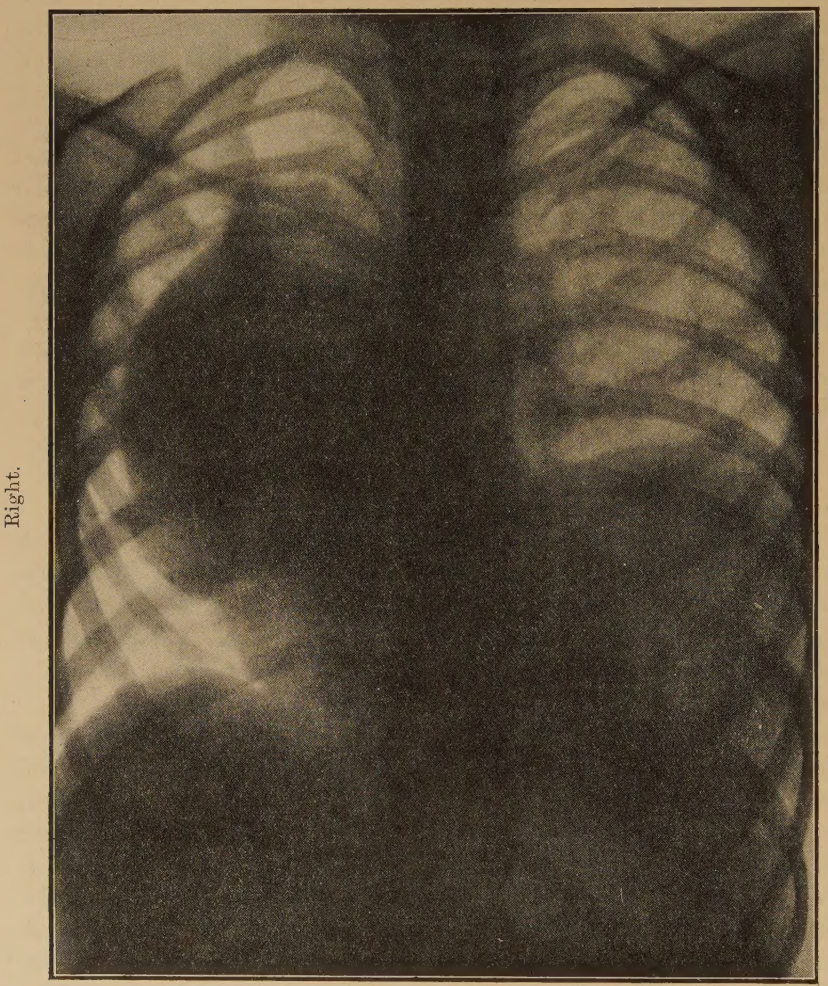

Fig. 1.—Skiagram of a boy, aged 5½ years, showing the shadow of an aneurysm of the ascending aorta on the right, and of a diaphragmatic hernia on the left.

attacks of sickness. The usual complaint, however, was the cough. Three months after leaving the hospital the gurgling sounds which the mother had noted were very distinct. A radiographic plate (see Fig. 1) was taken. The shadow on the left was interpreted

as being due to a diaphragmatic hernia. The clinical note was made, at the same time, of impairment of resonance and diminished breath-sounds over the left lower lobe.

During the year and five months that the child was under observation no change in the cardiac condition was detected. There was always dyspnœa on exertion, yet his general health continued good. He was seen two weeks before his death.

In March, 1917, while in an ordinary state of health, and with no unusual exertion, while undressing to go to bed he suddenly cried out, and fell over dead.

Post-mortem Examination.

One of us was permitted to make a partial examination in the child's home.

Thorax.—On opening the chest the pericardial sac, much distended with fluid, occupied the greater part of the cavity. Neither adhesions nor fluid were present in the pleural cavities. The pericardial sac with the heart was removed without opening for later examination.

Lungs.—There was complete collapse of the right middle lobe and partial collapse at both bases. The larger bronchi showed a chronic catarrhal condition, otherwise the lungs were normal.

Abdomen.—The colon was dilated. The stomach was dilated, and lay nearly entirely within the thoracic cavity. The diaphragm was distinctly thinner on the left than the right side. The œsophageal opening admitted the four fingers easily, and the dilated cardiac portion of the stomach passed readily through this opening into the thoracic cavity.

Except for fœtal lobulation of the kidneys, the liver, spleen and kidneys showed nothing abnormal.

Examination of the Heart.

On opening the pericardium about 16 oz. of clotted blood escaped. There was no pericarditis. A fusiform aneurysm of the ascending arch of the aorta, in size equal to the heart itself, presented. On the posterior surface of this aneurysm was a linear rupture of 2 cm. The auricles were not dilated. The endocardium was smooth and glistening, and all valves were normal in appearance. No degenerative changes in either myocardium or endocardium were noted. There was a small opening between the two auricles.

Except for this defect, and the aneurysm, the heart appeared quite healthy.

Prof. S. G. Shattock has examined the specimen of aortic aneurysm, and has kindly supplied us with the following detailed report:

"The ascending portion of the arch of the aorta is widely and uniformly dilated, so as to form a fusiform aneurysm which has a diameter approximately of 4 cm. and a length of 10 cm. The wall of the aneurysm is notably thickened, its measurement being 3 mm., whilst the thickness of the thoracic aorta is only 1·5 mm., and that of the trunk of the pulmonary artery also 1·5 mm., as taken after preservation in Kaiserling's fluid. The aorta beyond the aneurysm appears small by contrast; its diameter beyond the origin of the main trunks is ·7 cm. The ductus arteriosus is normally obliterated. In the posterior wall of the aneurysm there is an oblique rent, 2 cm. in length, which opened into the sac of the pericardium.

"The auriculo-ventricular, aortic and pulmonary valves are normal; there is a small communication between the auricles.

"The wall of the left ventricle is hypertrophied.

"The aorta is stenosed, but not closed, between the insertion of the ductus arteriosus and the origin of the left subclavian artery.

"Microscopic examination of the wall of the aneurysm shows that the thickening is due to a hyaline sclerosis affecting the deeper part of the intima and the immediately subjacent zone of the media.

"There is no lymphocytic or polymorphonuclear leucocytic infiltration. The cells, which are somewhat scantily distributed in the thickened intima, are of the connective-tissue kind; they are much increased in size and vacuolated, and are devoid of any regular disposition; they are of various form, and lie in distinct spaces in the dense hyaline material. In the subjacent portion of the media the muscle cells retain their parallel arrangement in the sclerotic basis.

"The fibrous nature of the thickening is shown by its staining red with Van Gieson's picro-fuchsin.

"As told by the use of Scharlach, the vacuolation of the cells is due to the accumulation of fat; in a few instances the cytoplasm contains one drop of fat, but in most, numerous and somewhat coarse droplets. None of the fat has been set free by actual disintegration of the cells.

"In sections stained with orcein a certain amount of elastic tissue persists in the hyaline material, throughout which it has been disparted in disconnected fragments; whether it is diminished in actual amount is difficult to determine.

"In sections stained with carbol thionine no bacteria are shown, and no spirochætes were discoverable in sections stained by Levaditi's

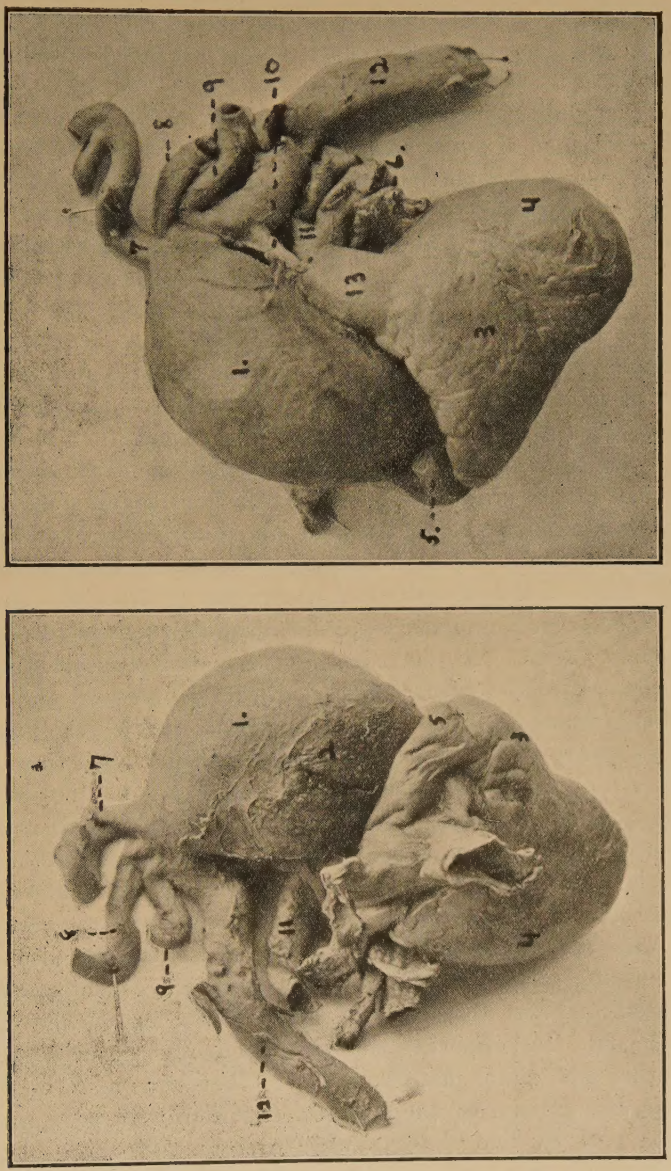

Fig. 2.—1. Ascending aorta, showing aneurysmal dilatation. 2. Linear rupture. 3. Right ventricle. 4. Left ventricle. 5. Right auricle. 6. Left auricle. 7. Innominate artery. 8. Left carotid. 9. Left subclavian. 10. Ductus arteriosus. 11. Left pulmonary artery. 12. Descending aorta. 13. Pulmonary artery.

method; although numerous pseudo-forms arise from the staining of the fine elastic fibres, these want the proper undulation.

"The most probable view, perhaps, of the pathogenesis of the condition is that, in consequence of the increased tension of the arch on the proximal side of the stenosis, a general protective sclerosis of the intima has been set up, the subjacent portion of the media being also involved; this has proceeded *pari passu* with the dilation, a vicious circle being established.

"Against a syphilitic origin are: The absence of the Wassermann reaction; the absence of spirochætes in the tissue; the strict limitation of the disease to the portion of the aorta subject to abnormal pressure, and the absence of any involvement of the aortic valve; the uniformity of the change, and the want of small cell-infiltration."

Discussion.

The necropsy revealed the cause of sudden death and also the explanation of the clinical signs referable to the heart. During life the diagnosis was not clear. There was a large pulsating mass in the thorax to the right of the sternum, while to the left of the sternum the heart seemed to be either displaced or enlarged, as judged by the position of the apex, 1 in. beyond the nipple line. Examination of the left side of the heart revealed nothing abnormal. On the other hand, to the right of the sternum and as far out as the right nipple there was a loud systolic murmur, present also, although not so loud, in the pulmonary area. To the pulsating mass on the right of the sternum was ascribed the paroxysmal cough, from pressure, and also the venous engorgement of the veins in the neck. With the exception of the shortness of breath on exertion, which persisted throughout his life, no symptoms were directly referable to the heart. A diagnosis of aneurysm was strongly suggested by the skiagram, and in the case of an adult one would not have hesitated to make this diagnosis with confidence. On the other hand, no evidence of the presence of any disease likely to be associated with aneurysmal dilatation was apparent, and apart from disease the condition is extremely rare in early life. The other diagnosis considered was that of congenital heart disease, with great dilatation of the right side of the heart. In support of this view there was the presence of numerous other congenital malformations. There was also the definite cyanosis, brought on by the slightest exertion, with accompanying dyspnœa. If cardiac changes were present they were clearly congenital in origin, for there were no evidences of acquired heart disease. The final diagnosis was not made until the necropsy.

A condition of diaphragmatic hernia was not at first suspected.

Early in the case it was noted that there was some impairment in the percussion-note at the base of the left lung, and that the air-entry there was deficient. This was a variable condition and might be altered by placing the boy on his right side. An early skiagram showed a shadow external to the cardiac area on the left side. It was not until the mother referred to the "gurgling" sounds produced by the boy, and which could be felt and heard, that the possibility of diaphragmatic hernia was considered. Another skiagram showed a definite shadow in the left side of the thorax, and a diagnosis of diaphragmatic hernia was made. This diagnosis explained the attacks of pain in the chest associated with vomiting which the mother had described. The necropsy showed that while nearly the whole of the stomach had passed into the thorax, the width of the opening was such as to allow movement of the stomach and its contents.

We have to express our great indebtedness to Prof. A. Keith and Prof. S. G. Shattock for their assistance and advice. The specimen of the aortic aneurysm described is now in the Museum of the Royal College of Surgeons.

We consider this aneurysm to have been due to a congenital defect in the development of the aorta. This conclusion is strengthened by the presence of other congenital defects—the shape of the head, ears, and eyes, the subluxation of joints, and a diaphragmatic hernia—and by the absence of any inflammatory changes in either heart or aorta. A partial stenosis between the insertion of the ductus arteriosus and the origin of the left subclavian artery had resulted in a fusiform dilatation on the proximal side in an otherwise healthy aorta.

Aneurysms similar in origin to the above have been reported by Smith and Targett, and by Wasastjerna (see below).

Ætiology.

Aneurysms in children may be classified according to ætiology into—(1) Arteriosclerotic; (2) traumatic; (3) embolic or septic; (4) false, due to erosion from without; (5) due to congenital anomalies.

(1) Aneurysms due to sclerotic and atheromatous changes in the aorta and the great vessels have been much studied in recent years. Among the earlier reports of aneurysms in children, Moutard-Martin, Sanné, and Berry have mentioned the presence of atheromatous patches. Discussions of this cause of aneurysms are given by

Oppenheimer, Willson and Marcy, Chiray and Ségard, Moizard and Roy, Rebaudi, Franckel, Veale and Carey Coombs, and v. Koós. In regard to the ætiology of the degenerative changes, evidence of lues is often present. Microscopic, as well as macroscopic study of the aortas from stillborn infants, has revealed infiltration with mononuclears, the presence of spirochætes, and early degenerative changes in a surprising number of cases. Such lesions in infancy suggest that many aneurysms of adolescent and early adult life may be due to congenital lues. For instance, Nixon has reported an aneurysm of the abdominal aorta associated with an extreme grade of atheromatous degeneration in a girl, aged 20 years, who showed no clinical signs of congenital lues except Hutchinson's teeth.

(2) True traumatic aneurysm of the aorta is exceedingly rare in childhood. We found in the literature only the doubtful case reported by Pendin.

(3) Under embolic and septic aneurysms are included those due directly to a septic embolus, as in the cases of Jordan and Voelcker, also the cases associated with rheumatism, as described by Comby, Marfan, Zuber, and Moore. De La Rue and Feytaud regard as rheumatic several instances with definite atheromatous changes in the aorta.

(4) An aortic aneurysm in a child due to an erosion from without has been reported by Hutchinson. The aneurysm below the constriction in Smith and Targett's patients, and the aneurysm described by Willet, both probably belong to this group. J. Jackson Clarke's cases are reported very briefly. In the first a caseous bronchial gland had discharged itself into both the aorta and the trachea; in the second, a child, aged 3 years, an aneurysm appeared to follow erosion by an inflamed lymph-gland.

(5) Since our case comes under the group due to congenital malformations, we shall give brief summaries of the recorded cases of this type.

(1) (*Martin*, 1827).—In an infant, aged 1 month, an aneurysm of the ductus arteriosus, the size of a small nut, and filled with blood-clot, was found.

(2) (*Thoma*, 1890).—In a woman, aged 26 years, who died of typhoid fever, there was an aneurysm of the aorta at the point of insertion of the ductus arteriosus. An exceedingly short ductus was partially closed toward the pulmonary artery side by a circular membrane, but opened like a funnel into the aorta, with no dividing line between ductus and aorta. Thoma considered the origin of the aneurysmal dilatation to be the traction of a much-shortened and

unobliterated ductus on the aorta. Dr. John Thomson described a case of congenital heart disease in a female child, aged 4½ years, in whom the ductus arteriosus was about ¼ in. long, with a small aperture into the pulmonary artery, a similar ring of intima, and a cone-shaped widening toward the aorta. Apparently his case was a less conspicuous example of the same type of dilatation.

(3) (*Smith and Targett*, 1897).—A boy, aged 9 years, was admitted to hospital, and died soon after tracheotomy was performed. At the necropsy an aneurysm of the arch of the aorta just beyond the origin of the left subclavian artery was found. About 1 in. distal to the dilated arch, and exactly opposite the attachment of the ductus arteriosus, the lumen of the aorta was suddenly narrowed by means of a septum with a central oval aperture, which measured $\frac{3}{16}$ in. in its chief diameter. The dilated portion of the aorta had thin walls, and most of the elastic tissue was lacking. There was nothing abnormal about the adventitia or endothelium. Immediately beyond the septum the aorta resumed its normal size, and ½ in. beyond the mouth of an aneurysmal sac was reached. This aneurysm was oval, 1½ by 1 in. in diameter. It was sacculated, and the coats of the vessel could not be followed into the sac. The tissues about the aneurysm were infiltrated with inflammatory products, so that the œsophagus, aneurysm and aorta were matted together.

It is easy to explain the fusiform aneurysm proximal to the stenosis. The sacculated aneurysm distal to the stricture was probably due to erosion of the aorta from without as noted above.

(4 and 5) (*Roeder*, 1900).—In a monograph on the subject, two cases of rupture of aneurysms of the ductus arteriosus were reported. In the first, a male infant, dying on the second day of life, the aneurysm was fusiform in type; in the second, a female infant, dying on the third day, a dissecting aneurysm had ruptured. In neither case was there any suspicion of syphilis.

(6) (*Wasastjerna*, 1903).—A healthy boy, aged 13 years, died suddenly. The walls of the aorta were exceedingly thin and the aorta dilated. A rupture had occurred not far from the semilunar valves. The endocardium, valves, intima and coronary arteries were normal. Just below the origin of the subclavian artery there was a nearly complete congenital stricture of the aorta. A remarkable collateral circulation had been established.

(7) (*Dry*, 1916).—A congenitally weak infant died on the third day of life. Between the pulmonary artery and aorta was a fluctuating, cystic mass the size of a large cherry. It contained a small

amount of recent blood-clot. There was no evidence of thrombosis. Communication was free into both pulmonary artery and aorta. No luetic history was obtained, and no signs of this disease were found about the child.

Phänomenow's case of abdominal aneurysm as a cause of dystocia we think was probably of syphilitic origin, as fatty degenerative changes were found in the aorta.

There are thus on record five aneurysms associated with congenital anomalies of the ductus arteriosus, and three cases, including our own, of aneurysmal dilatation of the thoracic aorta associated with congenital stricture of the aorta.

Ruptured Aortic Aneurysms.

In the literature we have found seven cases of ruptured thoracic aortic aneurysms, as follows:

(1) (*T. R. Smyth*, 1834).—An apparently healthy boy, aged 16 years, was seized with sharp pain in the region of the heart after ascending a ladder. He died fifty-one hours later. At the necropsy the pericardium contained 36 oz. of blood. The aortic valves were cartilaginous and considerably puckered. The mitral valves were somewhat thickened. There was cardiac hypertrophy, especially of the left ventricle. Death was due to the rupture of an aneurysm, the size of a walnut, on the arch of the aorta.

(2) (*Jones*, after Crisp, 1847).—An aneurysm of the ascending aorta in an athletic boy, aged 16 years, ruptured into the pericardium.

(3) (*Berry*, 1898).—A healthy boy, aged 15 years, fell over dead when about to bowl at cricket. The pericardium was distended with 12 oz. of blood. The ascending aorta for the first inch was closely adherent to the pericardium. Just below the line of reflection of the pericardium there was a transverse tear in the aorta, and below this, a fusiform aneurysm. In the wall of the sac and at the point of rupture were a few fatty patches. Thinning of the media was obvious. In the descending aorta there were a few small patches of atheroma. The aortic valves were slightly thickened. The ventricles were normal. Berry found no signs either of congenital syphilis or rheumatism.

(4) (*Jordan*, 1903).—A male child, aged 6½ years, who had suffered from otorrhœa since babyhood, was taken acutely ill with fever and vomiting. He was very ill for four days, and then gradually improved. Two weeks later, when apparently convalescent, he died suddenly. At the necropsy there was an ante-mortem clot in the left lateral sinus. The pericardium was full of blood. On the

ascending arch of the aorta was a sacculated swelling with a horizontal slit-like rupture. A triangular flap opened outward from the aorta into this tumour. The aorta, cardiac muscle, and the valves were normal. Jordan thought the origin of the aneurysm was in a localised septic aortitis, possibly from a minute embolus in one of the vasa vasorum.

(5) (*Wasastjerna*, 1903).—This case has been described under aneurysms due to congenital malformations. Wasastjerna classed his case as a ruptured aorta, but the dilatation, shown by his sketch and by his description, is very similar to that in Smith and Targett's and our own cases.

(6) (*Horder*, 1907).—A boy, aged 12 years, was admitted to St. Bartholomew's Hospital with pain in the chest. He had marked cardiac enlargement to the left. Nine days after admission he had a spasm of difficult breathing and died in a few seconds. The pericardium contained 12 oz. of blood. The heart was small and almost empty. The valves, myocardium, and coronary arteries were normal. The first part of the aorta was elongated and greatly dilated, forming a large fusiform aneurysm. The fusiform swelling ceased rather abruptly above at the level of the ductus arteriosus. Considerable endarteritis was present, with nodular thickenings of the intima, but without ulceration. About one inch above the aortic orifice the inner and middle coats were torn by a transverse rent, and another rent extended upwards. Toward the pulmonary artery the ruptured portion was covered by a fresh clot, and led into a dissecting aneurysm the size of a small walnut. A hole in this dissecting aneurysm indicated the point of rupture into the pericardium. There were no definite signs of congenital syphilis or rheumatism. The dissecting aneurysm probably originated at the same time as the onset of the pain, two weeks before death.

(7) (*Willson and Marcy*, 1907).—A male child, aged $3\frac{1}{2}$ years, had been under observation since six weeks old, when a loud systolic murmur heard best at the aortic area, but transmitted over the entire chest, was noted. When three years old he had a febrile attack of unknown origin, otherwise he had been fairly healthy. At $3\frac{3}{12}$ years he showed marked general glandular enlargement. Over the episternal notch there was expansile pulsation, and a thrill was palpable over the entire upper chest, posteriorly as well as anteriorly. The percussion note was flat over the manubrium, and to right and left for $2\frac{1}{2}$ in. There was a loud, almost continuous, systolic-diastolic bruit, with its greatest intensity over the second interspace to the right of the sternum. Both the aortic and

pulmonic heart-sounds were diminished. The radial pulses were equal. The arterial tension was high and the radial arteries thickened. One week after these notes were made he was taken much worse, with occasional rigors. The left radial pulse could no longer be felt. Death occurred two days after the onset of the rigors.

At the necropsy the heart was in systole. An aneurysm of the ascending and transverse arch of the aorta had ruptured into the pericardium, through an ulcerated area 1 cm. in width. Considerable pericarditis along the upper and outer area of the visceral and parietal pericardium was present. The aortic valves were much stenosed, the opening almost occluded, and the aorta showed extensive patches of atheroma. The authors regarded the case as undoubtedly one of syphilitic origin.

Closely related to these seven cases of rupture of thoracic aortic aneurysms is the following case of ruptured aorta:

(*Oppenheimer*, 1905).—A girl, aged 9 years, who was usually healthy, complained of headache and fainted. Two days later she fell over dead. At the necropsy a defect was found in the aorta just above the semilunar valves, with rupture at this point. The rupture started in the intima and media, and involved the adventitia at one point only. The media of the aorta was diseased, showing fatty, and, in places, atheromatous changes. Even in parts macroscopically normal, microscopically media, intima, and elastic tissue fibres showed pathological changes. No other signs of syphilis were noted, and there was no arterial sclerosis elsewhere.

Two instances of ruptured abdominal aortic aneurysms have been described:

(1) (*Miguel*, 1834).—A male child, aged 14 years, was admitted to hospital in a collapsed condition, and died the same day of a ruptured aneurysm of the abdominal aorta. Two other similar aneurysmal tumours were found. There was no disease of the heart.

(2) (*Armitage*, 1857).—A girl, aged 7½ years, with a history of palpitation all her life, was first seen five weeks before death. She had increased præcordial dulness and hypertrophied cardiac action. A few days before death she complained of pain in her joints. While sitting up one day she had a convulsion, followed in fifteen minutes by a second. She died an hour later. At the necropsy there were neither endocarditis nor atheromatous changes in the coats of the arteries. The aorta had ruptured at its bifurcation. The intervening space was a sac the size of a walnut, with thin, ragged

walls. On the posterior wall of the right iliac artery was a small patch of diseased intima.

The history in MacKeen's and also in Renon's cases (as described by Feytaud) strongly suggests a ruptured thoracic aneurysm as the cause of death, but no post-mortem examination was permitted in either. We have omitted descriptions of aneurysms, as those of Rie, Smith, Roger, Zuber, Barbier, Comby, Le Boutillier, Bacelli, and MacNalty, diagnosed clinically, but not confirmed by post-mortem examination.

Summary.

(1) A case is recorded of death due to the rupture of a fusiform aneurysm of the ascending arch of the thoracic aorta in a child, aged $5\frac{10}{12}$ years. The aneurysm apparently followed a partial stenosis of the aorta between the insertion of the ductus arteriosus and the left subclavian artery. That this stricture was a congenital anomaly was supported by the presence of a diaphragmatic hernia, subluxation of the joints, and defective cranial development.

(2) A brief discussion of the ætiology of aneurysms in children is given.

(3) From the literature are summarised two instances of aneurysmal dilatation due to partial stenosis of the thoracic aorta, also five cases of aneurysms apparently due to congenital anomalies in the structure of the ductus arteriosus. In two of the latter, rupture caused death.

(4) In the literature we have found seven reports of death following rupture of the thoracic aortic aneurysms in children, one case of rupture of the aorta without aneurysm, and two cases of rupture of aneurysms of the abdominal aorta.

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THE "FOOD DEFICIENCY" OR "VITAMINE" THEORY IN ITS APPLICATION TO INFANTILE BERI-BERI.

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The problems of nutrition are daily assuming a wider significance, and no aspect of the question has a greater interest than that of the part played by the "accessory food factors," or vitamines, as, following Funk's somewhat premature and misleading nomenclature, they have been called.

From the well-known observations of Hopkins to the recent work of Cole and Jordan Lloyd all research tends to emphasise the extensive *rôle* these unknown substances play in the growth and nutrition of the organism from the lowest to the highest forms.

It is therefore important that in the case of beri-beri, in which the accessory factor finds its most concrete application in clinical medicine, we should endeavour to determine and define the exact function of the specific vitamine concerned. For while the practical problem of the prevention of beri-beri may be considered to have been largely solved by the work of Chick and Hume (5), who have given precision to our knowledge of the distribution of the vitamines concerned, and have rendered possible the evaluation of a given diet in respect of its vitamine content and its beri-beri producing potentialities, yet the physical and structural chemistry, and hence the mode of action of these bodies, remain entirely unknown.

Although occurring in Europeans under certain conditions of dietary limitation, it is among those oriental races of whose diet highly milled rice forms the staple article that the disease is most common. Infantile beri-beri occurs among the children of the latter, and has been defined (9) as "an acute or subacute disease affecting infants who are nursing mothers suffering from beri-beri. It is characterised by cardiac hypertrophy and a peripheral neuritis, and results in a clinical picture of generalised œdema, dyspnœa,

cardiac disturbance, gastro-intestinal derangements, oliguria and aphonia. It is a form of true beri-beri, and like the beri-beri of adults is caused by the deficiency of some important substance in the food—that is, in the milk from a mother who is suffering from a similar deficiency."

Although on clinical and pathological grounds both Japanese and Philippine observers—to whom, with the Americans, we owe our knowledge of the disease—have regarded it as an intoxication, yet Andrews (1) and Chamberlain and Vedder (4) believe that their demonstration of a "deficiency" element in its production excludes this possibility. Whether both factors may not be operative is a point it is proposed to discuss.

While infantile beri-beri cannot be said to constitute a problem of directly practical importance in England, yet in respect of the light it throws on the adult form of the disease, and on the ætiology of the deficiency diseases in general, it has an interest of its own.

The Present Position of the Vitamine Theory.

From avian and human experiments and observations independently it has been concluded that beri-beri is directly due to the absence of a specific accessory factor, or vitamine, from the diet. This is supposed to be followed by an impaired metabolism of nervous tissues, and a degeneration of these that is the basis of the disease. Broadly stated, this is the vitamine, or deficiency theory of the origin of beri-beri. Whether this is adequate to account for all the facts, and to constitute a complete elucidation of them or not, it is the object of this paper to discuss.

In 1911, speaking of the function of the so-called "antineuritic vitamines," Fraser and Stanton (6) observed: "Whether these substances act by rendering other elements in the diet available for nutrition, or whether they are themselves the nutritive material necessary for nerve tissues, can in our present state of knowledge only be matter for conjecture."

From this point there has since occurred a divergence among subsequent workers in this field. Vedder (9) and his American co-workers hold that the antineuritic vitamine is directly concerned in the nutrition of nervous tissues, of which it is "an essential building stone." When a diet lacks this, the nutrition of the nervous system at once suffers, and it degenerates progressively until a point is reached at which the symptoms of beri-beri appear. According to this view, therefore, the disease consists of a slowly progressive,

diffuse, degenerative process affecting the entire nervous system (Vedder*).

A somewhat different view is held by Funk, Braddon and Cooper (2), and the majority of recent observers. They maintain that, in an as yet unknown way, the antineuritic vitamine is concerned in carbohydrate metabolism, and is essential to the organism in direct proportion to its carbohydrate consumption.

This hypothesis still lacks complete formulation, nor have its upholders followed it to its logical conclusion. For, if correct, it indicates the existence of a second factor in beri-beri production: not only is the absence of vitamine an essential condition, but the presence of carbohydrates in the diet must be equally so. To these as yet no *rôle* has been assigned, though they must manifestly play some part in the genesis of the disease according to this view. However, even the most recent work—for example, that of Chick and Hume—still describes "the vitamine whose presence in a diet is an essential for the proper nutrition of the nervous system." From which it appears that, although the association of vitamine with carbohydrate in this instance may be accepted, the deficiency theory as commonly held is still a purely negative, single-factor hypothesis, in which the profound significance of Braddon and Cooper's observations does not obtain recognition.

There are sufficient data for an analysis of these two views, and it seems clear that Vedder's hypothesis is untenable, while that associating the antineuritic vitamine with carbohydrates constitutes a distinct advance thereon.

The question may be approached from several aspects. We may ask, first, whether the clinical and pathological facts of beri-beri are compatible with the view that it is a slowly progressive diffuse

* It is not always easy to determine precisely what Vedder's views are, on account of the frequent and disconcerting modifications these undergo in the process of correlating fact with theory. Thus, in describing the symptomatology of beri-beri in his book (9), he states that the motor disturbances are due to a paralysis which is a "direct result of the neuritis." Later we read that "in fowls at least the paralysis is not caused by the neuritis. It is extremely probable that the same is true of human beri-beri," and again, "peripheral neuritis *per se* cannot be the essential lesion of beri-beri," and finally, "the paralytic symptoms are particularly caused by the degenerative changes in the cells of the cord and possibly the brain." Thus he establishes the thesis, which his theory necessarily involves, that the nervous system is the seat of a diffuse, progressive degeneration due to vitamine starvation. Apart from the neurologically unsound character of these propositions and of the data upon which they are based, the rationalising process of which they give evidence must react unfavourably on the weight that can be attached to work so dominated by speculative considerations.

nervous system degeneration; secondly, whether deprivation of vitamines is alone sufficient to produce it; and thirdly, whether carbohydrates can be excluded as a causal factor in its production.

Elsewhere (11) I have endeavoured to demonstrate that the clinical characters of beri-beri are incompatible with Vedder's explanation of the pathogenesis of the disease. The fulminating character of acute pernicious beri-beri, in which a robust man in the full vigour of life, after an acute illness of two or three days, dies a more or less agonising death of cardiac failure (Hamilton Wright (7)), the clinical identity of the neuritis of beri-beri with other forms of toxic polyneuritis, and the cardiovascular phenomena, cannot but convince the neurologist that the theory is totally inadequate to account for the facts.

Further, from the earliest pathological investigations of Baelz, Scheube, and Pekelharing and Winkler to the very recent observations of Lhermitte (8), there has been a consensus of opinion among writers that the nervous lesion is not specific for beri-beri, and is indistinguishable from that of a toxic polyneuritis. The last-named observer is very clear on this point, and he concludes his observations as follows: "La constation des lésions corticales est un trait de plus qui permét de rapprocher la polynévrite alimentaire avitaminique, le béri-béri experimental, des polynévrites toxiques." These conclusions of a writer, unique among investigators of the disease in that he is a neuropathologist, are most significant in their bearing on the disease.

Judged by the clinico-pathological method, which exponents of the deficiency theory consistently ignore, all the facts of the disease indicate an intoxication with a special, but by no means exclusive, incidence in the nervous system.

The symptom-complex and pathological basis of beri-beri are thus seen to be very striking and positive phenomena, quite unlike the slow dissolution of structure and function of the nervous system implied in Vedder's theory, and it may be asked whether a purely *negative* factor, such as the *absence* of a given food constituent or vitamine, can be conceived as the sole, direct and immediate *cause* of a disease in which polyneuritis, to mention but a single aspect of the disease, is a predominating symptom.

It is not too far-fetched, perhaps, to recall in this connection a famous axiom of Hughlings Jackson with regard to cerebral disease. He said that negative or destructive lesions could not cause positive symptoms, but might allow of their development—in other words, two factors were concerned in the production of a spastic paralysis,

destruction of pyramidal fibres causing the negative symptom paralysis, and a consequent unbalanced activity of other centres causing the positive symptom spacticity.

So with beri-beri, *absence* of vitamine cannot be an adequate *cause* of polyneuritis, which is clearly a positive reaction to a direct and positive cause. A carbohydrate metabolism, disordered in consequence of this privation, might, as we shall see later, conceivably provide this direct and positive cause.

The neglect of this principle is very evident in all writings on the deficiency theory of the origin of disease, and in the case of beri-beri is the obstacle before which the theory is at present halted. Its application to all presumed deficiency diseases is no less worthy of consideration.

There is considerable evidence in support of this *à priori* reasoning on both clinical and experimental sides, and this may be briefly resumed. Fraser and Stanton (6) found that fowls, fed on water only, outlived the latent period necessary for the appearance of polyneuritis in appropriately fed birds, dying eventually in the fourth and fifth weeks with neither clinical nor pathological signs of the disease. On the other hand, Chamberlain and Vedder (3) record the appearance of microscopical evidence of nerve degeneration as early as the seventh day in fowls on a white rice diet. From this it follows that simple deprivation of vitamine—and in this respect starvation is the equivalent of an avitaminic diet—does not suffice to cause polyneuritis. A second, positive factor is necessary.

It is apparent from all recent experimental work that this second factor is apparent by carbohydrates. In the recent outbreaks of beri-beri, particularly in the classic feeding experiment of Fraser and Stanton on coolies, as I have pointed out elsewhere, this factor is very manifest. There seems no unequivocal evidence that human beri-beri or the analogous polyneuritis gallinarum occurs in its absence. Maurer, Caspari and Moszkowski, Funk, Cooper, and Braddon and Cooper conjointly have all given convincing proof of this. Braddon and Cooper find that the physiological requirements of the experimental birds in the matter of vitamine vary directly with their carbohydrate consumption and not with the body-weight. Eijkman's early experiments with starvation rations of white rice foreshadowed this result.

In a recent paper (1918) Vedder re-states his view that beri-beri is a direct vitamine-starvation degeneration of the entire nervous system, and expresses the belief that the antineuritic vitamine enters into no association with carbohydrates.

He objects that in the experiments of Funk and of Braddon and Cooper the birds did not die of polyneuritis, but were killed by the effects of forced feeding with rice. Further, he claims to have produced polyneuritis in fowls by means of a carbohydrate-free diet, and to have shown that the addition of polished rice to such an avitaminic diet can both defer and prevent polyneuritis.

If these conclusions were sound, not only would they entail the rejection of the accumulated evidence quoted above, but they would also destroy the whole basis of the deficiency theory. For if the addition of polished rice to an avitaminic dietary can, by virtue of the improved carbohydrate-protein balance thus obtained, defer or prevent the development of polyneuritis, as he states is the case, it follows that vitamines are not essential constituents of a well-balanced diet, and the fabric of the deficiency theory falls to the ground.

It is scarcely surprising that a consideration of the experiments of Funk and of Braddon and Cooper affords no confirmation of the suggestion that death was due to forced feeding. The most that can be said is that in a few instances this mechanical factor may have hastened death in birds in whom there was clear evidence of polyneuritis in course of development. In the case of Funk's observations the objection does not apply at all. Moreover Vedder's own observations gave a similar result, for he records that his fowls were "too weak to stand up," while post mortem "a certain degree of degeneration was found by the Marchi method" in the nerves.

Of his meat, egg, and combined meat-rice and egg-rice feeding experiments it may briefly be recorded that they appear mutually contradictory in their results. If meat or egg rendered avitaminic by sterilisation at 120° C. produces polyneuritis, and if, as we know it does, polished rice also produces it, it is somewhat difficult to credit that a combination of the two "actually deferred the onset of polyneuritis," or in some cases even prevented it.

The only possible explanation of these discordant results is that the meat and egg did not produce polyneuritis, were not vitamine-free, and thus averted the onset of neuritis when combined with the polished rice. We know that egg is very rich in the active principle, and there is abundant reason for doubting whether heating for half an hour in an autoclave registering a temperature of 120° C. is adequate to destroy this completely.

The present state of our knowledge may therefore be summarised as follows: Beri-beri is dietary in origin in that it is associated with a physiologically inadequate diet. Yet the purely negative vitamine-

starvation explanation of its pathogenesis, apart from the inadmissible nature of its contained assumption that a disease which clinico-pathologically is clearly an intoxication could be *solely and directly* caused by the mere *absence* of anything from the food, is inadequate in that it does not recognise the existence of the two factors essential to the production of the disease. These two factors are—(1) the absence of an unknown "accessory factor," or vitamine; and (2) the consumption of carbohydrates. The recognition of these renders the non-production of beri-beri by starvation comprehensible. The fundamental error in the vitamine theory is an untenable conception of the neuropathology of the disease. We may therefore state the pathogenesis of beri-beri thus: the use of certain foodstuffs (carbohydrates) in the absence of certain specific and unknown accessory factors or vitamines directly causes beri-beri, though in a manner as yet undetermined.

Infantile Beri-Beri.

In the study of infantile beri-beri we may regard the problem from another aspect. As in the adult, the great majority of observers who have considered the disease primarily from the clinico-pathological standpoint have regarded it as an intoxication due to an unknown poison ingested in the maternal milk.

From their accounts it manifests itself as an acute and rapidly fatal convulsive malady in previously healthy infants of from one to three months of age.

In the less frequent chronic form are seen obstinate vomiting, constipation, progressive pallor and weakness, evidence of cardiac disorder, oliguria, and equivocal signs of neuritis. Unless the child is removed from the breast death results. At autopsy are found generalised œdema, serous effusions, hypertrophy, and dilatation of the right heart, marked passive congestion of the organs, and some peripheral nerve degeneration.

Applied to this picture, Vedder's theory of a diffuse, slowly progressive nervous system degeneration due to vitamine starvation is acutely incongruous.

For according to this we have to assume that during the months of intrauterine life, when the nervous system is being built up in the fœtus, and after birth, when myelinisation and development are proceeding apace, there is a shortage of the vitamine necessary for the nutrition of the nervous system—a lack of "something essential for the growth and development of the child's nerves" (Andrews(1)).

In these hypothetical circumstances a well-nourished child is actually born with a normally functioning nervous system, and after some weeks of normal life is suddenly stricken with an acute illness and dies in a few hours, unless vitamine-containing elements are at once added to its feeds, when within a few days the child is normal again (4).

We may well ask how it is possible, in such conditions of intra-uterine life and development (for a vitamine-starved mother can no more supply the essential principle to her fœtus than to her suckling), that a nervous system of normal structure and function can be laid down and reach completion, and how it can be conceived that a nervous system suffering from the time of its inception in the womb from a consistent starvation of an "essential building stone" can be rendered normal within the compass of a single week by feeding the child with an extract of rice bran (4).

It is not necessary to labour the point, for it is obvious that in its application to infantile beri-beri in this form we have the *reductio ad absurdum* of the deficiency theory.

The objections urged by the disciples of the deficiency theory to the clinico-pathological indications that beri-beri is an intoxication seem chiefly based on the failure to find toxic bodies in polished rice. It is also implied that since deficiency of vitamine is the necessary condition of its production, therefore beri-beri cannot be an intoxication. Leaving this *non sequitur* for the moment, we may consider the more material objection that no chemical or microbic poison has ever been isolated from a beri-beri-producing rice, and that the milk of the beri-beric mother cannot contain a poison causing the disease in the suckling, since the simple addition of the antineuritic vitamine to the child's diet is sufficient to cure it, and because "it is most irrational to believe that such an extract of rice polishings could cure a child in a few days while it was still receiving the toxin which had originally produced the condition" (Vedder).

That the polished rice diet of the oriental, as eaten, contains no poison has been clearly established. Nevertheless, the intervention of a second factor in the causation of the disease, and the recognition of carbohydrates as constituting this, make it quite possible that we may yet be dealing with an intoxication.

I have suggested (11) that the problem may be considered in the following way: Since the antineuritic vitamine exercises its functions in association with carbohydrates, and since its absence from a dietary in which these predominate results in the profound patho-

logical changes that we recognise as beri-beri, it is not unreasonable to suppose that in these circumstances carbohydrate metabolism may at some stage become disordered, and that this disorder results in the production of some toxic bye- or end-product which is the direct cause of beri-beri.

Such a view is necessarily speculative. It has, however, the following merits: it is compatible with the view associating beri-beri with dietary deficiency, it elucidates the hitherto obscure relationship of general starvation to vitamine deprivation, and it accounts for the failure of starvation to produce experimental beri-beri.

It recognises the two factors co-operating in the production of beri-beri, and assigns to each its proper place in the ætiology. It is compatible with the clinico-pathological phenomena of the disease, and finally, it reconciles the older toxæmic with the newer deficiency theory. In its present form the latter satisfies no one of these postulates and rejects both the true and the false of its predecessors. Like them, it reveals upon investigation its own modicum of error.

Having assigned a function to carbohydrates in the ætiology of beri-beri, it is interesting to speculate further from what is known of the nature of the antineuritic vitamine as to its mode of action.

The physical chemistry of this substance has not been fully investigated, but such facts as we possess indicate the possibility of its being an enzyme. It is relatively thermolabile, it is inactivated in the presence of alkalies, and it forms adsorption compounds. No element can be said to be an integral part of it. The more the active principle is purified, the fewer characteristic reactions does it show and the more unstable it becomes. It is now recognised that it has never been isolated. Whether it exists in the colloidial state or not is not known. Its resources—namely yeast, the eggs of animals, and the embryos of seeds—are fruitful sources of enzymes. Indeed, from yeast is derived in abundance the enzyme glyoxalase, which is thought to be essential to carbohydrate hydrolysis in the body.

Possibly the vitamine under discussion is a substance of this nature and stands in a comparable relation to carbohydrates. In its absence, it being a complex the body cannot synthesise; carbohydrate breakdown may be arrested at some point short of complete hydrolysis, leaving a toxic product in circulation.

In this respect infantile beri-beri presents no discordant phenomena. The abnormal toxic metabolite we have assumed to exist in the organism in adult beri-beri, passing to the infant in the maternal

milk, produces the disease. The addition of the active principle in the rice bran extract will cause its complete hydrolysis, and render it innocuous in the child just as in the adult.

While it is infinitely more probable that a woman suffering from a mild chronic toxæmia can give birth to a normal child at full term than that one whose system lacks an essential constituent or food of nervous tissues to such an extent that her own nervous system is slowly degenerating should do so, yet it would be interesting to know the frequency of miscarriages and stillbirths among beri-beric mothers.

It is clear that much remains to be done before the deficiency diseases can be regarded as completely elucidated, and it is difficult to see how the purely negative vitamine-starvation theory can be correlated with the clinical and pathological phenomena of beri-beri.

The physical and structural chemistry of the accessory factor involved, the exact nature of its association with carbohydrates and the part played by the latter still remain an unexplored field. The accessory food factor is the child of the physiological laboratory, and despite the advances made by the investigators of beri-beri, it is, in all probability, from the physiological chemist that we shall learn its nature and its mode of action.

In the meantime, a return to a more purely clinico-pathological outlook on beri-beri than is at present adopted by students of the disease should prove corrective of a tendency, distinctly noticeable in many writings on the subject, to adapt ascertained phenomena of the disease to the procrustean bed of what is at best but an immature theory.

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ON ABNORMAL NAUGHTINESS IN NORMAL CHILDREN.

By John Thomson, M.D., F.R.C.P.Edin.,

Consulting Physician to the Royal Edinburgh Hospital for Sick Children.

Occasionally a child of perhaps 3 or 4 years or older is brought to the doctor because he is becoming so persistently and enterprisingly naughty that his parents have begun to doubt whether he is altogether right in his mind. He is generally found to be healthy and attractive-looking and above the average in intelligence as well as in force of character, although we are given such a bad account of his conduct. We are told that the child is constantly doing wilful mischief—throwing china out of the window, knocking over flower-glasses, scattering beads or other things that are difficult to pick up again, breaking in upon other children's games so as to spoil their play, locking doors and throwing away the keys, pulling out drawers and emptying their contents on the floor, setting fire to anything that will burn, playing unkind tricks on the cat, or even upsetting the baby out of the cradle. The account of the child's misdoings is rather amusing, and one notices that, in spite of the anxiety which has occasioned the consultation, the parents describe them with just a touch of pride, and a feeling that not every child would have the energy and cleverness to be so entertainingly naughty.

When we inquire into the treatment that has been tried, we usually find that, until recently, the child's mischief has been laughed at and rather encouraged. Since the parents have thought more seriously about the matter, however, the young culprit has been subjected to a course of severe corporal punishments, reprimands and expostulations; and it has been attempted to make him ashamed of himself by relating his misdeeds to friends and neighbours in his presence with severe comments. These measures have not caused the slightest improvement.

On considering the child's conduct, we see that the things he has

been doing were calculated to give him little or no pleasure in themselves, yet they were a very real source of gratification because of the sensation they produced among his seniors. The effect of his misdeeds in disturbing his parents is, then, the source of their attractiveness to him ; and so great is the child's enjoyment of this that the beatings and other unpleasant experiences that followed have counted for nothing against it. They have been taken indeed merely as evidence of the greatness of the impression he has succeeded in producing.

What is wrong with the child in such cases is a morbid exaggeration and perversion of the natural desire which all children feel for notice by their elders. He craves to be the most noticed person in the household, and the obvious though unexpressed admiration provoked by his boldness and resource is all the encouragement he needs to encourage him to continue.

It is, I think, clear that these cases of abnormal naughtiness in normal children are of essentially the same nature as those of "moral imbecility" in children who are in some degree mentally defective. The treatment of the two sets of cases is the same, but the mental defect in the latter group makes their treatment at home much more difficult, if not impossible.

The proper lines of treatment are clear enough, and when carried out consistently and thoroughly are rapidly and completely successful. They may be summarised as follows: (1) All severe corporal punishment must be stopped—it never does any good. (2) Nobody must ever appear shocked, amused or even surprised at anything the child does. (3) His misdeeds are never to be alluded to, much less described, in his presence ; and, lastly, he should be noticed and encouraged in every way when he is good, and altogether ignored when he is naughty.

CASE OF MUSCULAR SPASM IN A CHILD.*

By Reginald C. Jewesbury, M.D., F.R.C.P.,

Assistant Physician to the Victoria Hospital for Children and Charing Cross Hospital.

With Remarks by F. Parkes Weber, M.D., F.R.C.P.

The rarity of the following case is sufficient to warrant its being placed on record:

The child, a girl, aged 9 years, of British nationality, had enjoyed

* The case was shown at the Section on the Study of Disease in Children of the Royal Society of Medicine on May the 24th, 1918.

perfect health until she was 7 years of age, when she began to suffer

Spasm of left arm and legs.

Showing talipes and spasm of shoulder muscles.

Showing torticollis.

from weakness and wasting in the muscles of both legs. This condition gradually progressed, and she developed such a marked degree

of talipes equino-varus that double tenotomy of the tendo Achillis was performed at a provincial hospital. After this she was able to walk better for a time, but the condition gradually recurred and the legs became very stiff when she attempted to use them, and walking became exceedingly difficult. Subsequently, about eighteen months after the legs began to be affected, she developed a similar loss of power with stiffness in the muscles of both arms and hands, and also in the muscles of the neck and spine. The child was brought up to my out-patients at the Victoria Hospital in April last, about two years after the onset of her complaint.

On examination she was a healthy-looking girl, normally developed and highly intelligent. There was no history of any previous illness, with the exception of measles. Both her parents were English; the father had suffered from asthma and died of pneumonia. There were three other children in the family and all quite healthy. The patient had lived in a healthy country place all her life.

There was marked weakness and wasting of the muscles of both legs with a double talipes equino-varus; she was unable to put her heels to the ground, and walked on tip-toe with curious jerky movements of the legs but not a typical spastic gait. Both arms were similarly affected, but less so than the legs. Any attempt to carry out a definite movement with the arms was made in a jerky way, rather like a child suffering from chorea. In grasping any object all the voluntary muscles of the body were thrown into a state of spasm. She appeared to have a certain amount of control over this spasm, and it was always much more marked when she was at all excited or agitated. She also had a persistent torticollis with a certain amount of spasm of the spinal muscles. The facial muscles were unaffected. No tremors or athetosis were present.

The reflexes, both superficial and deep, were all normal; the knee-jerks were only difficult to obtain when the muscles were in a state of spasm. The plantar reflex was flexor on both sides. The cranial nerves were normal. Sensation was unaffected.

The blood gave a negative Wassermann test.

The photographs give some idea of the condition described.

The child remained in hospital for about three months, and was treated with massage without any appreciable improvement.

Remarks by Dr. Parkes Weber.

The case seems to be an example—perhaps the first recorded in England—of "Dystonia musculorum deformans" (Oppenheim) or "progressive torsion spasm of childhood." Other names have been

given to the disease by various observers, such as "toxic torsion neurosis," "general tic with toxic changes," "tortipelvis." Ziehen's account dates from 1910. Opinions have differed as to whether the condition is organic or functional. H. Oppenheim, however, who described the disease in 1911 as "a peculiar spasm-like disease of childhood and youth," and suggested as a name for it Dysbasia lordotica progressiva or Dystonia musculorum progressiva, regarded it as an organic affection closely allied to athetosis.

Like paralysis agitans, Huntington's chorea, progressiva athetosis, pseudo-sclerosis, and Kinnier Wilson's progressive lenticular degeneration, it has lately been attributed to disease of the lenticular nucleus. It runs at first a chronic progressive course, with intermissions and even periods of improvement, and may then come to a standstill and remain stationary, other portions of the nervous system remaining unaffected. Though essentially a disease of childhood, it has appeared as late as the seventeenth year. It is characterised by a peculiar mixture of hypertonus and hypotonus of the affected muscles. The face and muscles of articulation are usually not affected except in late stages, when there is involvement of the muscles of the neck. Amongst the earliest symptoms, or the earliest symptom, is partial or total loss of function in one or more extremities—usually the lower extremities—due to faulty tonus innervation. Talipes tends to persist. The movements of the affected parts are spasmodic but not truly athetotic. Psychical symptoms may occur, but the mental condition is generally normal. As yet, the disease has been described only in Russian, Polish, and Galician Hebrew families, but the present case seems, in this respect, to be an exception.

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Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Friday, April the 26th, 1918.

Dr. H. Morley Fletcher, *President, in the Chair.*

Case for Diagnosis.—Dr. H. C. Cameron and Mr. W. H. Trethowan.—The patient, a boy, aged 5 years, was born at full term and was breast-fed. There were six children in the family. All the others were in good health. There had been no deaths and no miscarriages. The father and mother were healthy. Some difference between the two sides of the body were noticed at birth. The left arm and leg were considerably shorter than the right. The boy used both hands and feet, and was able to walk and run, though with some limp, on account of genu valgum on the left side. The phalanges of the left hand were shortened, and were broader and flatter than those of the right. The little finger showed a permanent flexion which could not be reduced. The right arm and leg were apparently normal and well formed. The right little finger only showed a slight deformity in its proximal phalange. There appeared to be no abnormality of the nervous and vascular supply to the affected limbs. The Wassermann test was negative. No other abnormalities were found. X-ray examination showed surprisingly advanced changes in the bones of the affected limbs, especially in the femur and hand. The epiphyses were normal throughout. The diaphysis of the phalanges was distended into a mushroom-shaped expansion, with its convex surface towards the distal end of the bone. The metatarsals showed a similar expansion. The outline of the femur was in many places indistinct, and the shadow was mottled by irregular areas of lighter colour. The only abnormality seen in the skiagram on the right side of the body was in the little finger, where changes similar but of earlier development were seen. No abnormality of the skull, ribs, scapula, or pelvic bones was found.

Dr. Parkes Weber regarded the case as an atypical one of multiple enchondromata.

Perithelioma of the Orbit.—Mr. H. R. Jeremy. A girl, aged 4 years, was admitted to hospital on April the 14th, 1917, with the diagnosis of "dermoid of orbit." The right eye had been growing out during the past three months, and the child had screamed with pain in the eye, forehead, and temple. No history of injury or vomiting. Family history good. On admission the right eye was proptosed and displaced downward and outward. Muscular movements of the eye were restricted upwards and inwards. The conjunctival vessels were congested, and there was chemosis below. The cornea was clear, the iris reacted, and the pupils were equal. Examination of the fundus showed that the vessels were full and tortuous, the disc clean, and of normal colour. Under the upper and inner margin of the orbit a soft lobalised tumour could be felt, not adherent to skin, and movable from side to side, but attached deeply. The surface of the tumour was

smooth and elastic. There were no enlarged glands, and there was nothing abnormal in the chest or abdomen.

Operation.—A 2 in. incision through the base of the upper lid exposed a soft white tumour, which was encapsulated; it consisted of two lobes united by a stalk, which was attached to the optic nerve behind. The stalk was ligatured close to the nerve, and the two lobes removed. The tumour was friable and ruptured during removal. There were slight adhesions between the capsule and surrounding structures. The wound was dried and closed with silkworm sutures. The child contracted scarlet fever during the following week and was removed to a fever hospital. She was readmitted two months later with recurrence of the tumour, which had now forced the eye so far forwards that it could not be covered by the lids. As the result of exposure the cornea had ulcerated and ruptured and the iris had prolapsed through the corneal wound. The surface of the tumour was ulcerating. As it was useless to try to save the eye, Mr. Jeremy removed it and cleared out the entire contents of the orbit, including the periosteum. The child had remained well since the operation and had increased in weight. Section of the tumour showed it to be a perithelioma.

Discussion on Botulism.—Dr. H. Morley Fletcher said that he had recently seen two cases resembling the recently recorded adult cases of botulism. The first case was in a girl, aged 2 years, who ten days previously to being seen by Dr. Morley Fletcher had been getting very sleepy. She would fall asleep even over her meals, and it became increasingly difficult to rouse her. The onset was quite gradual, and she did not suffer in any way. On admission to hospital, in addition to her drowsiness, she presented double ptosis and left external strabismus. The right pupil was larger than the left. The reflexes were normal, and there was no fever. Early tuberculous meningitis was diagnosed. Lumbar puncture showed no increase in cells beyond what a slight admixture of blood would account for. The somnolence gradually passed off in about a fortnight after admission and the ptosis and squint disappeared. The second case, which occurred in a boy, aged 3 years, was almost identical. He had a history of falling asleep at meals and being in a very drowsy condition. Like the girl, he was well nourished, and but for the sleepiness seemed well. He had ptosis and a slight squint and his condition cleared up in about a fortnight.

Dr. J. D. Rolleston suggested polioencephalitis as an alternative diagnosis. Judging from the literature, it was not so much the cerebrum as the fons and medulla which were affected in botulism, and it was possible that the two cases were due to the unknown virus of encephalitis.

Dr. J. Porter Parkinson and the President subsequently described cases in adults.

Friday, May the 24th, 1918.

A Case of Anæmia with Unusual Blood-count.—Dr. J. Porter Parkinson.—A boy, aged 9 years, was admitted to hospital on March the 24th. Six weeks previously bruising all over the arms, shins, and back was noticed, and, a week later, a rash resembling flea-bites had appeared on the body, and had been coming out at intervals since. For a month he had been bleeding at the mouth, the lips were painful and swollen, and the gums sore. For a fortnight he had had severe headache, and was noticed to be rather pale and breathless. He had had scarlet fever at 18 months, "kidney

trouble and dropsy" a year later, measles and whooping-cough when 6 years old, but no other illness. There was one other child, aged 3 years, who was healthy. No family history of hæmophilia. On admission he was very pale and his face rather puffy. There was no rash except a few petechiæ on the feet. The gums were not swollen, but were bleeding slightly. The tonsils were enlarged, and a few glands could be felt in the neck. The lungs and heart were normal except for a pulmonary hæmic murmur. The liver and spleen appeared to be of nornal size. The urine contained neither albumin nor sugar. The temperature for the first fortnight rose to 99° F. in the evening, but in the last week had risen to 100° F. most evenings. The blood-count on April the 25th was: Red cells, 1,260,000; leucocytes, 800; Hb., 20 per cent.; colour index, 0·9; polymorphonuclears, 29 per cent.; lymphocytes, 72·5 per cent.; eosinophils, 0·5 per cent. No nucleated red corpuscles; no marrow-cells. The red corpuscles showed marked alteration in size and shape. May the 6th: Red cells, 1,088,000; leucocytes, 1200; Hb., 20 per cent.; colour index, 1; polymorphonuclears, 18 per cent.; lymphocytes, 76 per cent.; hyalines, 5 per cent.; eosinophils, 1 per cent. The red cells showed marked differences in size and slight differences in shape. No nucleated forms. The lymphocytes consisted almost entirely of small cells.

The cause of the profound anæmia was obscure. There was no reason to suspect scurvy, hæmophilia, tuberculosis, or syphilis. The Wassermann reaction in the blood was definitely negative. Lead-poisoning was unlikely. There was no evidence of intestinal parasites. The blood showed marked leukopenia, due almost entirely to diminution in the number of the polymorphonuclears, the lymphocytes being in relative excess but actually about normal in number.

Ophthalmoscopic examination showed slight optic neuritis and numerous hæmorrhages into both retinæ.

Dr. Parkes Weber regarded the case as a typical example of chronic aplastic anæmia, in which condition the red bone-marrow was the only tissue at fault. There was a deficiency of the blood-cells which were formed in the red bone-marrow—viz. the red corpuscles and the polymorphonuclear leucocytes. Practically nothing was known as to the cause of that type of case.

Case of Muscle Spasm.—Dr. R. C. Jewesbury (*vide* p. 269).

Abstracts from Current Literature.

Diseases of the Alimentary Canal.

Congenital paramedian sinuses of lower lip (*'Arch. of Ped.,'* 1917, xxxiv, *p.* 924).—**N. Toomey** reviews the literature, including the cases of Clogg (*v.* British Journal of Children's Diseases, 1907, iv, *p.* 52), and Kahn (*ibid.*, 1913, x, *p.* 127), and reports a case in a female infant, aged 8 months, with complete cleft of hard and soft palate: 3 mm. on each side of the median line of the lip, and 6 mm. orally from the muco-cutaneous border was a sinus, 5 mm. deep, which, when squeezed, gave out a small drop of clear mucus. J. D. Rolleston.

Perlèche (*'Journ. Amer. Med. Assoc.,'* 1917, LXIX, *p.* 192).—**J. E. Lane** describes perlèche as a superficial—not an ulcerative—streptococcic affection of the labial commissures, which is pre-eminently a disease of infancy and childhood. The epithelium becomes smooth, whitish, and later macerated, when small transverse fissures appear. It is not impetigo, but may be associated with it, and is not to be confused with herpes labialis, eczema, stomatitis, or syphilis, though it closely resembles a mucous patch. The disease is highly contagious, but generally can be promptly cured by painting the lesions with 10 per cent solution of silver nitrate, diluted tincture of iodine, or copper sulphate. If these fail a 5 per cent. solution of chromic acid is efficient.

T. R. WHIPHAM.

Perlèche: its bacteriology, symptoms and treatment in 223 cases (*'Arch. of Ped.,'* 1917, XXXIV, *p.* 274).—**A. L. Smith.**—Perlèche is an infection of the labial commissures, manifesting itself first by a maceration of the epithelium, secondly by a degeneration of the tissue, and thirdly by the formation of shallow ulcers and cracks. It is undoubtedly contagious, and is essentially a disease of children. An anaërobic streptococcus was isolated in 135 cases at an early stage of the disease. Twenty-six times it was found in combination with the *Staphylococcus pyogenes aureus*, 7 times with the *Staphylococcus albus*, and 14 times with the *Streptococcus pyogenes*. In the later stage the anaërobic bacillus was not found at all, but the *Staphylococcus pyogenes aureus* was found alone 36 times, and was associated 3 times with the *Streptococcus hæmolyticus*. Perlèche is more prevalent during the colder months, 73 per cent. of the cases occurring in the winter. It seems to be secondary to abnormal conditions about the oral and nasal cavities which cause an increased flow of saliva. It is usually bilateral. It must be distinguished from impetigo, which is more wide-spread and does not begin at the corners of the mouth, and from syphilis, in which the lesions are deeper and more wide-spread, are copper-coloured, and usually affect the mucous membrane inside the mouth, where perlèche rarely appears. The Wassermann and luetin tests will usually decide. Treatment consists in painting the lesions with 50 per cent. silver nitrate solution, and when dry applying Lassar's zinc oxide paste. If the lesions do not disappear in a few days, spirits of camphor or alcohol are applied. If the flow of saliva does not decrease in amount, tincture of belladonna should be given internally until the physiological limit is reached (*cf.* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1908, V, *p.* 459).

J. D. ROLLESTON.

Rumination in the first year of life (*'Amer. Journ. Dis. Child.,'* 1917, XIV, *p.* 210).—**C. G. Grulee** reviews the literature, and records six cases observed in a period of three years, including that of Stransky (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1916, XIII, *p.* 246). He is therefore inclined to think the condition is much more frequent than is ordinarily supposed, and that it can be explained on the basis of a hyperexcitability of the involuntary circular muscles as suggested by Hess (*ibid.*, 1915, XII, *p.* 214). The cases may be divided into two types—the mild and the severe. In the mild types the rumination occurs only once or twice a day, and is not accompanied by any definite loss of food. In severe cases the attacks are very frequent and a large proportion of food may be lost. Probably about 50 per cent. of the severe cases die. Treatment was directed along three lines: (1) Drug treatment: Bromides and other sedatives were

given, as well as cocaine and other local sedatives for the stomach. (2) Dietetic treatment: Thick vegetable *purée* or cereal gruel was of value in some cases, but in others failed. (3) Psychical treatment: All excitement should be avoided, the child should be in screened surroundings, but after food should have its attention diverted by playthings. J. D. Rolleston.

Pylorospasm and congenital pyloric stenosis (*'Amer. Journ. Dis. Child.,'* 1918, xv, *p.* 351).—**J. Foote** states that probably the earliest reported case of this condition appears in a text-book published by the English pædiatrist, George Armstrong, in 1777, in his text-book entitled 'An Account of the Diseases most incident to Children till the Age of Puberty.' Clinical and post-mortem findings, a typical case-history and a family history of three similar cases led Armstrong to conclude that the child aged 3 weeks, which he had treated for "watery gripes," died from "spasm of the pylorus." J. D. Rolleston.

Infantile pyloric stenosis (*'New York Med. Journ.,'* 1917, cv, *p.* 1251). —**J. Epstein** considers the view that this is a congenital malformation more likely than that it is acquired, as the latter would not account for the unusual rapidity of the hypertrophy. In addition, there is often a spasm of the pylorus which tightens the opening still further. The symptoms usually begin between the third and sixth week of life, with forcible, projectile vomiting and steady loss of weight. There is constipation and diminished amount of urine. Gastric peristalsis is usually seen after a meal, and the pylorus may be felt as a small, hard mass. Pylorospasm may be hard to differentiate, but it usually occurs in older children, and there is frequently a history of neurosis in the family. Medical treatment should always be tried first, consisting of proper feeding and lavage. Opium, atropine, and alkalies have been recommended. In surgery Rammstedt's operation is giving the best results, which consists in making an incision along the length of the pylorus down to the mucous membrane.

J. Porter Parkinson.

Congenital atresia of small intestine (*'Arch. of Ped.,'* 1918, xxxv, *p.* 45).—**F. V. Bogert,** who had previously published three cases of this condition (*v.* British Journal of Children's Diseases, 1917, xiv, *p.* 207), now adds a fourth. The baby was a well-developed child, born at full term. There had been three previous pregnancies, all resulting in healthy children. The abdomen became distended, and frequent vomiting occurred on the evening of the first day. The vomit became fæcal on the third day. Repeated enemata resulted in several small mucous curds and a large mucous cast of the bowel. Death took place on the fourth day. The necropsy showed complete obstruction of the small bowel ending abruptly 25 in. above the ileo-cæcal valve. There was complete absence of gut for about ½ in., and both small and large bowel below were poorly developed. No fibrous cord intervened between the two portions of gut.

J. D. Rolleston.

Congenital intestinal obstruction (*'Amer. Journ. Dis. Child.,'* 1918, xv, *p.* 354).—**A. V. StGeorge** reviews the literature, and reports a case in a female child, the second of twins, who died on the seventh day of life with symptoms of intestinal obstruction and hæmorrhagic disease of the newborn. The necropsy showed complete stricture of the duodenum, the lumen of which was replaced by fibrous tissue and a stenosis 20 cm.

below in the jejunum, the lumen of which was markedly diminished in calibre and occupied by a small blood-clot. J. D. Rolleston.

The big, flabby belly in infancy (*'Med. Press,'* 1918, I, *p.* 224).—**Marfan** calls attention to the big, flabby belly in infancy which is generally associated with rickets. In some instances the symptoms of rickets strike the eye, but in others they have to be looked for. Diagnosis will, however, not be complete until the cause—toxic or infective—of the rickets is established. In most cases hereditary syphilis, or dyspepsia, or tuberculosis is responsible, but to these must be added prolonged suppuration, eczema, etc., subacute recurrent broncho-pneumonia (as in whooping-cough), malaria, and diabetes mellitus. The big, flabby belly does not disappear for many months, even under the influence of appropriate treatment, because it follows the course of the osseous lesions. When the latter disappear then the belly resumes its normal shape. J. Allan.

Intestinal atony in children (*'New York State Journ. Med.,'* 1918, XVIII, *p.* 1).—**F. B. Turck** discusses the mechanism of production, and points out that there are a number of factors involved, as: (1) Increased permeability of the gastro-intestinal tract; (2) agencies responsible for this increased permeability; (3) a previous sensitisation of the muscle-cells, rendering them susceptible to anaphylactic reaction; (4) fatigue; and (5) tension. Other pathological states also exert an influence. In the treatment of severe acute cases where there is evidence of stasis and death is impending, the first indication is for heat stimulation, such as a hot sitz bath, as hot as can well be borne, and, in addition, gastric and colonic lavage with bicarbonate solution given at a temperature of 110–118° F. The lavage may with advantage be given continuously in severe cases. Starvation must be enforced and milk avoided by all means. One may administer subcutaneous injections of salt solution or of sugar in solution, just sufficient to create stimulation but not to burden the heart. Transfusions of blood are indicated in certain cases. After the acute stage has passed, feeding may be resumed gradually. Corn-starch gruel is highly satisfactory, but it is important to remember that all fats must be avoided. In the treatment of moderately severe or chronic cases gastric or colonic lavage should hold an important place, and should be given in connection with gentle pneumatic gymnastics of the colon. It is important to lengthen the period between feedings as much as possible, so as to afford rest to the gastric and intestinal musculature. Meat extractives should be eschewed. In older children extract-free meat, which should also be fat-free, may be allowed in moderation. On recovery the child may be given neutral fats in the form of fresh butter, olive or cotton-seed oil. Autogenous vaccines are beneficial in chronic cases. Drug treatment consists in the administration of the insoluble colloids, such as Irish moss and bismuth, or of fine bran and charcoal. For the treatment of acidosis mixed alkalis are indicated.

J. Allan.

Acute ileo-colitis in infancy (*'Arch. of Ped.,'* 1917, XXXIV, *p.* 504).—**J. Aikman** writes on 54 cases of ileo-colitis, 27 of which were under six months of age, 8 between six and twelve, and 12 between one and two. Only 4 were breast-fed. Insanitary home conditions, poverty, and general carelessness in food preparation were ætiological factors. Of the 27 children under six months, 13, or nearly half, died, while the total mortality was

33·3 per cent. The duration of the disease averaged fifteen days. The stools were mostly green, liquid, offensive, and all but 11 contained mucus. Blood was found in 28 cases; vomiting was present in 30 cases, and was very marked in one-fifth of the cases. Convulsions occurred in only 4 cases, all of which died. The temperature varied, but in 29 cases was never above 101° F. The physical signs were merely the same as those found in a case of malnutrition. Treatment: If the diarrhœa has been present for a short time, 1 or 2 drachms of castor oil should be given. If there is vomiting, calomel in $\frac{1}{10}$-gr. doses should be given. Milk should be omitted from the diet and cereal water with or without sugar given. Bismuth subnitrate in 10-gr. doses may be of value. Hot applications to the abdomen and hot mustard baths are frequently used. Subcutaneous injections of normal saline or sodium bicarbonate solution replace the loss of fluid. Local treatment consists in high rectal irrigations of a gallon or more of normal saline or a 1 per cent. sodium bicarbonate solution once or twice daily. J. D. Rolleston.

The cœliac disease (*'Lancet,'* 1918, II, *pp.* 163, 193, 227).—**G. F. Still** states that 17 cases occurred among 14,800 admissions to the Great Ormond Street Hospital. It is commoner in private and consultant practice, and therefore in all probability does not depend on poverty or defective sanitation. It is commoner among girls than among boys. In the present series of 41 as many as 30 were females. The onset is usually between the ages of 9 months and 2 years, and lasts for months or years. Clinically, the patients are emaciated, with plump faces and distended abdomen, much behindhand in general development, and afflicted with looseness of the bowels complicated by exacerbations of diarrhœa. The stools resemble porridge, are acid, rancid, and float in water. As the disease progresses, the stools become pale, greenish or grey. Vomiting, pain or fever are usually absent. The urine is normal. Pallor is due to a deficiency of hæmoglobin from failure of fat absorption. Although there is marked arrest of growth in stature as well as weight, there is no abnormality of the formed bone and no sign of rickets. The voice is of a high pitch and very limited compass—a striking contrast to the gruff voice of cretinism and mongolism. Dentition is delayed, but not markedly. There is great muscular debility; one case was unable to walk at 6 years old. Mentally the children are normal, but are slow in response. They are chiefly interested in their necessarily meagre diet. Œdema is the commonest complication, and is situated over hands, legs, feet, and lower eyelids. Occasionally there is ascites. Purpura was noted in several cases. Scurvy occurred in 5 cases. The disease strikingly resembles sprue; even chronic glossitis may occur in the cœliac disease. Only 5 of the cases in the 41 recovered completely and passed normal motions; on the other hand, only 6 died, and in 2 of these death was due to accidental complications. Pathologically there is marked thickening of the whole of the walls of the large and small intestine and stomach with patches of acute congestion of the mucosa. Microscopically there is marked round-cell infiltration. In 6 cases one or other or all of the types of dysenteric bacilli known as Flexner, Shiga and Y were discovered, either actually or by agglutination tests. Organisms for which a causal relation is claimed by Herter are known as the *B. bifidus* and *B. infantilis*. The bacteriological findings are not, however, conclusive. The stools were in three cases found to contain 26·9, 73·1, and 42·5 percentage of fat as compared with a normal of 10–25 per cent. There is evidence in some

cases of a failure of the fat-splitting ferments of the bile. The pancreatic function was normal in the majority of cases investigated by Sahli's, Loewi's and Stock's tests, and there is no failure in the splitting of fats. There is, therefore, a failure of fat absorption due perhaps to a catarrhal condition. The Wassermann reaction was tried in 6 cases with a positive result in 3. In none of these were there any symptoms of congenital disease, and in none were the sera of the relatives positive. The œdema is due in part to feeble circulation and hydræmia and in part to toxic absorption from the bowels. Tetany is also due to the same cause and not to increased elimination of calcium salts. With regard to the arrest of growth it is pointed out that increase in weight does not proceed *pari passu* with increase in height. It may be due to deficiency of fat absorption, as rats fed on lean, raw meat failed to grow. It is doubtful if this is due to deficiency of calcium, because in cretins exhibition of thyroid causes rapid growth, although there is relative deficiency of calcium salts. The fats must be reduced to a minimum. Dried milk is more easily digested than ordinary cow's milk. Cases do well on human milk, and no cases of cœliac disease have occurred when infants are being fed on the breast. Asses' milk is well borne, and this depends on some other factor than low percentage of fat, as babies with cœliac disease do not do well on cow's whey. The carbohydrate offered is not well tolerated, but that of lentils, commercially known as "revalenta," is generally suitable. Chicken broth, veal broth and sweet jellies complete the meagre diet. On such a diet as outlined above scurvy developed in 5 cases, which can be prevented by the addition of mashed potatoes. In spite of this fat-free diet there is marked immunity from rickets. Inunction of fats (*per cutem*) has been tried with doubtful results. Glycerine extract of pancreatic juice and the commercial products known as "trypsogen" and "holadin" and ox-gall were also exhibited with negative results. The most effective drugs are 5 minims of castor oil or 2 or 3 gr. of salol, which lessen the number of stools and improve their quality. Silver nitrate in doses of $\frac{1}{6}$ to $\frac{1}{4}$ gr. is also of use. Chromo-santonin in 2 gr. doses in gelatine capsules was also helpful. Iodoform was given with markedly beneficial effects in one case. As a considerable proportion of the cases gave evidence of intestinal infection with a dysentery bacillus an autogenous vaccine was tried, with temporary improvement in 2 cases. In 3 thyroid extract was tried, but without any result. Polyglandin was tried in a convalescent case, with striking and sudden increase in growth. CHRISTOPHER ROLLESTON.

Surgery.

Burns and scalds as a cause of child disablement and death (*'Dublin Journ. Med. Science,'* 1917, II, *p.* 248).—**Sir John Moore** read a paper on this subject at a meeting of the Royal Academy of Medicine in Ireland, Section of State Medicine. He submitted a statement, prepared for him by the Registrar-General for Ireland, showing the number of deaths from burns or scalds registered in the Dublin registration area during each of the five years 1912–1916. The total deaths from these causes of persons of all ages were 227, including 123 deaths of children under 5 years, the latter figure representing 54·2 per cent. of all these 227 deaths. Something more is wanted at coroners' inquests into fatal cases of burns and scalds among young children than a mere colourless verdict of "accidental death." The laws on the subject should be put into action. J. ALLAN.

Adenoma of the umbilicus (*'Brit. Med. Journ.,'* 1918, I, *p.* 564).—**C. H. Whiteford** describes a case in a boy, aged 5 years, who came under observation because of a red, smooth, oval tumour at the lower border of the umbilicus. The tumour was diagnosed to be an adenoma. It was excised, and the pathological report confirmed the diagnosis. The condition is one of the abnormalities which result from incomplete obliteration of the vitelline duct. J. ALLAN.

Congenital tumour in the fronto-nasal region (*'Edin. Med. Journ.,'* 1917, II, *p.* 383).—**D. M. Greig** describes a case in a male infant, aged 2 years. The child had also a large, double hare-lip and cleft palate, the cleft being much wider on the left than on the right side. Dependent from the region of the left fronto-nasal suture was a pyriform, thin-skinned, reddish tumour, 1½ in. in length. It had a translucent appearance, its surface was glazed and smooth, and it was of a somewhat soft consistency. The tumour could not be diminished by pressure, and there was no evidence that it communicated with the interior of the cranium. Under a general anæsthetic it was easily removed. The pathologist reported it to be "a congenital tumour composed of fibrous tissue, unstriped muscle, etc." J. ALLAN.

Congenital Dupuytren's contraction of the fingers (*'Edin. Med. Journ.,'* 1917, II, *p.* 384).—**D. M. Greig** records a case in a male infant, aged 5 weeks, who was breast-fed, well formed and well developed, and of good colour. The mother had noticed since birth a difficulty in getting the child's palms washed, in that she was unable to get the child's fingers extended. After careful examination under a general anæsthetic the author came to the conclusion that the condition was apparently not a tendinous affection, and there was no evidence of any abnormality in the growth or peculiarity of innervation of the muscles. It appeared to involve the skin, subcutaneous and deep fascia, and to be similar to the condition known as Dupuytren's contraction in the adult. J. ALLAN.

Multiple cartilaginous exostoses (*'Arch. of Ped.,'* 1917, XXXIV, *p.* 338).—**P. M. Stimson** reports a case in a boy, aged 9½ years, who was prematurely born at the time of his mother's menopause and following a severe nephritis during the last two months of her pregnancy. Irregularities in his bones were first noticed at the age of 4 years, since when they had grown slowly and become more numerous, finally involving all the long bones, the scapulæ, clavicles, and ilia. His extremities also showed a marked distal hypotonus. His mental age was that of a child aged 5 years, and the development of his carpal bones and some of his ossification centres about that of a child of 6 years. His mother and an older sister also had exostoses. J. D. ROLLESTON.

A case of multiple cartilaginous exostoses (*'Arch. Bras. de Med.,'* 1917, VII, *p.* 313).—**M. Junior.**—The patient was a boy, aged 14 years, who presented more than fifty cartilaginous exostoses, chiefly on the long bones, at the site of the epiphysial cartilages. The head was not affected. The first exostoses to appear were on the ribs, where they developed at the age of two years. There were no other cases in the family. Although the Wassermann reaction was slightly positive, there was no other sign of syphilis in the patient. J. D. ROLLESTON.

The pathology of the congenital funnel-shaped thorax (*'Riv. di Clin. Pediat.,'* 1917, xv, *p.* 595).—**R. Simonini** evolves an interesting theory concerning the causation of this condition. He does not admit the influence of rickets, which is absent in the majority of the cases, nor arrest of development or excessive shortness of the sternum, nor the theory of Kundmüller that the condition is due to abnormal softness of the bones. He says that in some cases, where there is marked difficulty in breathing, it is not rare to find a temporary funnel-shaped thorax due to forcible contraction of the diaphragm, so that not only the antero-inferior median parts become approximated, but also the lateral, corresponding to the insertions of this same muscle. He therefore thinks that the diaphragm plays an important part in the production of the funnel-shaped thorax. He moreover found in three autopsies that the liver was adherent to the diaphragm, and attributes the funnel shape of the thorax to its exaggerated and abnormal action under these conditions. VINCENT DICKINSON.

A case of cervical rib simulating Pott's disease (*'Arch. de méd. des enf.,'* 1918, xxi, *p.* 83).—**J. Renault** and **Romme** record a case in a girl, aged 13 years, who, owing to her habit of keeping her head and neck bent forward, had been wrongly supposed to be suffering from Pott's disease in the cervical region. Movements of flexion, extension and rotation all took place normally. The cervical column was normal, but the dorso-lumbar column showed some scoliosis. There was also some degree of lordosis. The right supraclavicular fossa presented a slight projection, which the X rays showed to be due to a cervical rib. The child also presented deformities of the fingers, extreme myopia, and a subnormal intelligence.
J. D. ROLLESTON.

Fractures of the neck of the femur in children (*'New York State Journ. Med.,'* 1917, xvii, *p.* 508).—**H. L. Taylor** reaches these conclusions: (1) Fracture of the neck of the femur may occur at any age. (2) The typical fracture of the neck of the femur in children up to thirteen is a hinge fracture at the junction of the neck and shaft; this fracture is one cause of coxa vara. (3) From thirteen to sixteen the typical injury is fracture at or near the epiphysial line, with marked displacement. (4) Above sixteen the type of fracture of the femoral neck does not differ from that in adults. Notes on seven cases are appended. J. ALLAN.

Fracture of the neck of the femur in a child (*'Journ. Amer. Med. Assoc.,'* 1918, lxx, *p.* 84).—**C. A. Parker** reports an instance of this rare fracture in children which occurred in a boy, aged 9 years, as the result of a fall from a height of 15 ft. The lesion was shown by a skiagram.
T. R. WHIPHAM.

A case of fracture of the pelvis with rupture of the bladder in an infant under 2 years, with recovery (*'Boston Med. and Surg. Journ.,'* 1918, clxxix, *p.* 100).—**R. W. Augevine.**—A female baby, aged 23 months, was run over by the rear wheel of an automobile. There were few symptoms of shock. Pain apparently was not great. The abdominal wall was slightly more tense than normal. X rays showed oblique fracture of the left ramus of the pubis. Bloody urine was removed by catheter. On laparotomy under ether free fluid containing blood and having a urinary odour was present in the abdominal cavity, and a tear $\frac{3}{4}$ in. long was found

extending antero-posteriorly along the bladder-wall. This was stitched, and a drainage-tube was left in the abdomen for forty-eight hours. Complete recovery took place. J. D. Rolleston.

A series of severe cases of osteomyelitis (*'Practitioner,'* 1918, ci, *p.* 163).—**R. A. Walker** describes six cases admitted to St. Thomas's Hospital in seven months. Case 1: A boy received a blow on the leg five weeks previously. Three days previous to admission the leg became painful. Numerous incisions were made. No pus was found, but a small amount of watery fluid was present in the tissues. Death occurred two days later, and the *Staphylococcus aureus* was present in the pus found in the pericardium and lungs. (2) Girl, aged 12 years: No history of trauma. Severe pain in the left foot and ankle, which were swollen. No pus was found on operation, which consisted of numerous incisions; as these did not improve the condition amputation below the knee was performed, but death occurred on the second day. Pus was found in the ankle-joint and sheath of the tendons, and contained the *Staphylococcus aureus*. (3) Boy, aged 15 years: Pain in the right leg and swelling as a result of a fall. On operation pus was found in the medullary cavity containing the same organism as in the other two cases. Recovery. (4) Boy, aged 8 years: No history of trauma to cause severe pain and swelling over the internal malleolus of the left leg. On operation pus was found in the medulla, and on the fifth day a pyæmic abscess formed on the chest-wall. Death occurred, and post-mortem examination revealed abscesses in the pericardium, lungs and kidneys. *Staphylococcus aureus* was found in the pus. (5) Boy, aged 4½ years: The left leg was swollen and very painful. There was a history of a slight fall six days previously. Subperiosteal resection of the shaft of the tibia was performed with success, but pleurisy with effusion requiring tapping occurred. *Staphylococcus aureus* was found in the pus and fluid. (6) Boy, aged 13 years: The right femur was painful, but there was no redness or œdema. A month previously he had a fall. An operation was performed. On the fifth day the left ankle became swollen and painful. The joint was opened and the pus removed. Death occurred. Post-mortem examination showed solid pneumonia in both lungs; no abscess. Pyæmic abscess in buttock. No pericarditis. *Staphylococcus aureus* was present in the pus.
F. R. B. Atkinson.

Salivary fistula following a simple mastoidectomy with cervical abscess (*'Ann. of Otology,'* 1917, xxvi, *p.* 113).—**F. C. Schreiber.**—A rare post-operative sequel. The case, a girl, aged 7 years, showed it in conjunction with facial paralysis—a double catastrophe regarded by the recorder as unique. She was operated upon for acute mastoid abscess with cervical abscess at the age of two. Macleod Yearsley.

The repair of cleft palate (*'Journ. Amer. Med. Assoc.,'* 1917, lxix, *p.* 1966).—**H. M. Sherman.**—All operators to-day are practically using the Langenbeck or the Lane method of closing the soft palate. The former, recognising that the cleft represents a failure of union, attempts to correct that fault by putting the parts in union with the least disturbance of tissues. The Lane method splits the velum between its pharyngeal and buccal sides, and turns flaps over from one side to the other. This necessarily jeopardises the palatal muscles in the splitting, and results in extensive scarring in healing. The Langenbeck method can be so done as to disturb the muscles individually not at all, though they are, as a

group, moved toward the middle line. The statement that the Langenbeck method is inadequate in a wide cleft with a flat palatal arch does not apply to the velum. Instead of paring the sides of the cleft to get raw surfaces for union, the author splits them from the attachment of the velum to the tips of the uvular halves, and sutures the posterior edges of the split uvular halves together, tying the knots in the pharynx, and follows by uniting the anterior edges, tying the knots in the mouth. Full details and diagrams of the author's operation are given in the text.

T. R. Whipham.

Congenital hypertrophic stenosis of the pylorus (*'Lancet,'* 1918, i, *p.* 804).—**T. J. Wood** reports a case in a male infant, aged 6 weeks, which was successfully treated by posterior gastro-enterostomy. The pylorus was in a very rigid state, and gastric lavage, as recommended by Hutchison, would probably have been of no service. J. Allan.

Hypertrophic pyloric stenosis in a two-year-old child (*'Journ. Amer. Med. Assoc.,'* 1918, lxx, *p.* 1913).—**A. J. Scott.**—Cases of hypertrophic pyloric stenosis after the second or third month are very rare. The present case was that of a boy, aged 22½ months, who was brought to hospital for vomiting, loss of weight and extreme restlessness which had set in about a month previously. The abdomen was scaphoid, with visible peristaltic waves but with no palpable tumour. A fluoroscopic examination showed that nothing passed through the pylorus. As the child's condition became worse in spite of stomach-washings, followed by gavage of barley-water and egg-albumin, operation was performed and revealed a typical pyloric stenosis. A modified pyloroplasty was carried out. Death took place the following day. J. D. Rolleston.

Intussusception in children (*'New York Med. Journ.,'* 1917, cvi, *p.* 400).—**G. Torrance** speaks of the importance of early diagnosis. Value should be attached to the mother's history of sudden screaming, vomiting, and afterwards occasional attacks of abdominal pain, with straining and blood passed *per rectum*. The only condition to be excluded is colitis, in which there is usually fever and frequent motions of mixed fæcal matter and bloody mucus. The abdominal tumour in intussusception is not always felt, and it is important not to wait till a tumour can be felt *per rectum*. One case of appendicitis and one of a spastic ileus have been reported simulating intussusception, but as the treatment was similar the mistake is of little importance. As regards ætiology, Laws says intussusception occurs more frequently in healthy, well-nourished infants; 49 out of 63 were under one year old. It has occurred as a sequel to whooping-cough and round-worms. Operation within the first twelve hours after the onset is the only treatment to be considered. J. Porter Parkinson.

Intussusception: its early recognition (*'Arch of Ped.,'* 1917, xxxiv, *p.* 781).—**H. Apfel** reviews the literature, and reports a case in a girl, aged 9½ months, who was operated on only nine hours after the first symptom. An ileocæcal intussusception about 12 in. long was found and reduced. Rapid recovery took place. J. D. Rolleston.

Two uncommon forms of intussusception occurring simultaneously in the same patient (*'Lancet,'* 1918, i, *p.* 437).—**J. A. C. Macewen** reports this case in a male infant, aged 18 months, who was admitted

to hospital with a history of persistent vomiting for three days prior to admission, and the passing of mucus, blood and slime by the bowel, but no fæcal matter. General condition was poor. At the operation a short intussusception of the small intestine about the upper end of the ileum was found. This appeared to be recent, was easily reduced, and involved about six inches of bowel in all. On further search a second intussusception was found at the splenic flexure, the transverse colon being projected into the descending colon for about three inches. This intussusception was less recent and was much more difficult to reduce. The infant recovered from the intussusceptions, but unfortunately developed a subacute pneumonia and died twenty-five days after the operation. J. Allan.

Intussusception of the bowel in an infant followed by a second intussusception three weeks later (*'Amer. Journ. Dis. Child.,'* 1917, xiv, *p.* 63).—**A. Peskind.**—The infant was not quite 5 months old. At the first operation the lower end of the ileum, appendix, cæcum, and part of the ascending colon were found telescoped into the transverse colon. The appendix, which was almost black, was tied off and removed. The rest of the incarcerated bowel was gangrenous in places, but the child's condition prevented resection. Within forty-eight hours the temperature and pulse became normal, and recovery seemed complete within nine days. Three weeks later, however, symptoms of acute obstruction developed and the abdomen was opened again, when several inches of the ascending colon were found telescoped into the transverse colon. This second intussusception was easily reduced, but the cæcum and part of the ileum at the site of the first intussusception were found matted together, and the lumen of the bowel seemed obliterated. Ileocæcostomy was performed, and though double pneumonia developed on the third day after the operation, complete recovery took place. J. D. Rolleston.

Acute intussusception: a rational method of treatment (*'Lancet,'* 1918, i, *p.* 803).—**S. W. Daw** was called upon to operate on a male infant, about a year old, for acute intussusception of some thirty-six hours' duration. The tumour was exposed and reduced up to the last few inches. Beyond that point no legitimate pressure would move it, and the bowel was beginning to show signs of giving way. The stage was reached at which resection is the usual procedure. By means of a pair of "ring forceps" the constricting entrance was enlarged, and the intussusception was then easily reduced. There was a little lymph on the portion of bowel thus exposed, but it appeared quite recoverable, so it was returned and the abdomen closed. The patient made an uninterrupted recovery.

J. Allan.

Chronic intussusception (*'Lancet,'* 1918, i, *p.* 215).—**H. W. G. Mackenzie** and **W. H. Battle** record a case in a boy, aged 15 years, who had complained simply of "noises in the stomach." There was no pain, no vomiting, no constipation. After careful examination a diagnosis of chronic obstruction of the lower end of the small intestine, caused by some mechanical change near the ileocæcal valve, was made. At the operation the ileocæcal angle, including cæcum and about 7 in. of the ileum, was resected. The parts removed showed the wall of the ileum to be nearly $\frac{1}{4}$ in. in thickness from hypertrophy. Water from a tap could not be forced along the lumen of the gut. When a longitudinal incision was made the condition was

shown as a long-standing intussusception, about 6 in. of the ileum being affected, and so closely adherent that the parts had to be separated with scissors. J. ALLAN.

An unusual case of malposition of the appendix (*'Journ. Amer. Med. Assoc.,'* 1918, LXX, *p.* 23).—**H. T. Miller** operated upon a boy, aged 4 years, for a mass over McBurney's point which was found to be the head of the cæcum. The appendix could not be found in the usual situation, nor any sign of it. In the mass, however, a smaller moveable mass could be defined with its base attached to the cæcum opposite the point usually occupied by the appendix. This the author concluded to be the diseased appendix. The case was left to nature and the abdomen was closed. On the fourth day there was a copious discharge of yellowish matter and blood, and later a yellowish-green slough about 2 in. long was passed, which was presumably the appendix. An uneventful recovery took place. T. R. WHIPHAM.

Appendicitis in infants (*'Arch. of Ped.,'* 1917, XXXIV, *p.* 641).—**I. A. Abt** has collected 80 cases of appendicitis in infants under two years of age, 20 of which occurred in infants under three months, 6 between three and six months, 11 between six and twelve months, and 40 from one to two years. Manifest chill, which is rare in childhood, is infrequent in the first stage of the disease. The temperature is unreliable, as it may be very high or subnormal. The pulse usually corresponds to the temperature. Constipation is the rule, and occurs among the more severe types. Diarrhœa occurs in the milder types, though both conditions may be present and alternate with one another. Hereditary predisposition to the disease is often noticed, probably owing to some peculiar structural weakness of the lymphoid tissue of the appendix. Traumatism occasionally plays a part in the production of appendicitis, though it is probable that it is only an exciting cause when there has been a pre-existing chronic appendicitis. The frequent association of bladder symptoms with appendicitis had been noticed by Van der Bogert (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1917, XIV, *p.* 208). As the appendix in childhood frequently lies deep, tenderness may be greater on the left than on the right side. Rectal examination is very important in such cases. Where there is palpable resistance on the right side in the presence of other symptoms, the diagnosis of appendicitis should be strongly suspected. J. D. ROLLESTON.

Abscess of the abdominal wall from intestinal perforation by worms (*'Giorn. di Med. Mil.,'* 1917, LXV, *p.* 1046).—**A. F. Canelli.**—A boy, aged 12 years, was admitted to hospital with a swelling the size of an orange in the ileocæcal region The abscess was opened, and on the ninth day a dead ascaris was found at the bottom of the cavity, and the next day more worms were found in a small quantity of liquid fæces. Recovery took place. J. D. ROLLESTON.

Abscess of the abdominal wall with discharge of twenty-three ascarides; recovery (*'Gazz. d. Osp.,'* 1918, XXXIX, *p.* 401).—**T. Fiori** records a case in a girl, aged 14 years, in which the abscess was situated about 6 cm. to the right of the umbilicus and about 1 cm. above a horizontal line passing through the umbilicus, in a region corresponding to the small intestine. The abscess opened spontaneously, and in the course of seven

months twenty-three worms were evacuated. The condition originated in an intestinal perforation, which had given rise to an adhesive peritonitis whereby the visceral layer became adherent to the parietal peritoneum.

J. D. Rolleston.

Ascaridiasis simulating appendicitis (*'Lancet,'* 1918, i, *p.* 637).—**A. Kidd** gives notes of a case in a female child, aged 8 years. The symptoms and physical signs pointed to appendix abscess and laparotomy was performed. The appendix was, however, normal and was not removed. At the operation the presence of round-worms in the intestines was detected. A course of treatment with santonin subsequently resulted in the evacuation of large numbers of round-worms at intervals, the total number being 212. **E. M. Walters** (*'Lancet,'* 1918, i, *p.* 725) reports a similar case in a child, aged 7 years. The total number of worms passed was 159.

J. Allan.

Death from volvulus caused by round-worms (*'Med. Record,'* 1918, xciv, *p.* 65).—**V. G. Heiser** reports a case in a child, aged 4 years. Two days after administration of oil of chenopodium the child developed abdominal distension with severe pains and frequent vomiting. Post mortem a mass of round-worms the size of a man's fist was found blocking the bowel; the intestines below the mass were so twisted as to cause complete obstruction. There was local peritonitis in the neighbourhood of the obstruction. More than 300 worms were found in the intestine.

J. D. Rolleston.

Notes on a case of strangulation of the cæcum in an inguinal hernial sac, complicated by acute appendicitis, in an infant, aged 16 months (*'Med. Journ. Austral.,'* 1917, ii, *p.* 5).—**H. Bullock** opened the sac and allowed the turbid fluid to escape, removed the appendix, freed the gut, and returned it to the abdominal cavity. Recovery was uneventful.

F. R. B. Atkinson.

Strangulated hernia in a child, aged 3 weeks (*'Med. Journ. Austral.,'* 1917, ii, *p.* 227).—**J. H. Ingham.**—Operation was successfully performed thirty-six hours after the onset of symptoms. F. R. B. Atkinson.

Umbilical fistula following empyema necessitatis (*'La Med. de los Niños,'* 1916, xvii, *p.* 7).—**M. Vargas.**—A boy, aged 6 years, was admitted to hospital with pus trickling from the umbilicus. There was a history of appendicitis, but a probe passed into the fistula did not lead downwards, but up under the ribs. Examination of the chest showed signs of extensive left pneumothorax. The seventh, sixth, fifth, and part of the fourth ribs were resected, together with part of the inferior angle of the scapula. Recovery took place, though the child stopped breathing during the removal of the sixth rib owing to pressure of the sternal end interfering with the heart's action. J. D. Rolleston.

Types of cerebral defects in children that may be benefited by operation (*'New York State Journ. Med.,'* 1916, xvi, *p.* 484).—**H. G. Matzinger** discusses what types of cases offer a reasonable hope of cure or sufficient benefit from operations upon the head to warrant operative interference. He thinks by far the most reliable and important sign of increased

intracranial pressure is changes in the fundus of the eye. These are easily discovered by ophthalmoscopic examination, and appear as congested vessels, choked disc, and atrophic changes, etc. Disorder of the cranial nerves also develops frequently and early. When these new symptoms appear in a case of infantile cerebral paralysis operation is indicated, and should be undertaken, not with the object of cure, but in the hope of prolonging life if such a result is desirable. J. Allan.

Double venereal infection in a child (*'Arch. Bras. de Med.,'* 1917, vii, *p.* 268).—**A. Farani.**—The patient was a girl, aged 6 years, infected with gonorrhœa and syphilis. Contamination was due to interfemoral coitus, which was performed by a man suffering from these two diseases under the belief that he would be cured by passing them on to a healthy subject. The child presented vulvo-urethritis, mucous tubercles round the anus, as well as fever, polyadenitis, and a general œdema due to acute nephritis, a rare complication of infantile syphilis. Considerable improvement followed specific treatment. J. D. Rolleston.

Orthopaedics.

The ætiology of Perthes' disease (*'Journ. Amer. Med. Assoc.,'* 1917, lxix, *p.* 1598).—**P. W. Roberts** states that the ætiology of osteochondritis of the hip, generally known as Perthes' disease, has been the subject of much speculation ever since the condition was recognised as a clinical entity several years ago. Traumatism, obscure infection and perverted metabolism have each had their advocates. Tuberculosis has been eliminated because the joints recover with good function, and syphilis has been excluded because Wassermann reactions have been negative. Notwithstanding the results of laboratory tests, the author advances the opinion that this affection of the hip, which occurs with considerable frequency in children, is the result of inherited syphilis. The negative Wassermann may be disposed of, for it is common experience that subjects with frankly syphilitic bone lesions, when a family history of syphilis exists and when the pathological process disappears under mixed treatment, give a negative Wassermann reaction. Therefore, it may be contended that a negative Wassermann reaction in Perthes' disease does not exclude syphilis. The pathological findings described by Perthes also coincide with one of the most common expressions of bone syphilis in children, namely, osteochondritis. Then, again, subjects with Perthes' disease show dental evidence of inherited syphilis. This does not mean Hutchinsonian teeth necessarily, but more frequently the erosions and malformations of the first permanent molars and other units that have been so well described by Cavallaro, or anomalies of spacing of the incisors. Finally, the course of osteochondritis of the hip is similar to that of many other syphilitic joint conditions in that the destructive process is self-limited, and that there is a tendency to more or less complete restoration of function. The author records two cases in girls, aged 8 and 11 years respectively, in whom the symptoms rapidly disappeared under specific treatment.
T. R. Whipham.

Treated and untreated osteo-chondritis juvenilis of the hip (*'Journ. Amer. Med. Assoc.,'* 1917, lxix, *p.* 1060).—**W. Blanchard** states that Perthes' disease or osteochondritis deformans juvenilis seems to be a nutritional disease of the bones and muscles of the hip and leg. The femoral head that continues in functional use during the acute stage of

the disease becomes obliterated wholly or in part by the attrition of weight-bearing on softened bone, while the head of the femur that is mechanically protected throughout the acute stage of the disease suffers only the same atrophic changes as the adjacent bones. In convalescence, the destroyed head re-develops rapidly under mechanical protection, and re-develops more slowly and less perfectly without protection. The disease progresses with one year of bone and muscle atrophy and lessened density followed by several years of increasing density and re-development. The bone hardening resembles the eburnation after rickets. From the foregoing facts it may be inferred that this disease of growing bone is nutritional in character, and is due to a distorted blood-circulation. T. R. Whipham.

The sphere of arthroplasty in the treatment of ankylosis (*'Edin. Med. Journ.,'* 1917, II, *p.* 176).—**Alexis Thomson** reports two cases in children. A girl, aged 14 years, was admitted to hospital on account of stiffness of the right shoulder. No history was obtainable. At the operation there was found an old-standing separation of the epiphysis and sub-coracoid dislocation of the head of the humerus. The greater part of the head was removed to secure free movement of the upper end of the shaft in the glenoid cavity; a portion of the fascia lata, large enough to wrap round the head of the bone, was removed from the right thigh and stitched in position between the bones; the limb was put up in the attitude of right-angled abduction. The object of the operation—a sufficient range of movement at the shoulder-joint—was attained. In the second case, a schoolboy, aged 13 years, had complete osseous ankylosis of the right knee following "acute arthritis of infants." Arthroplasty by Murphy's method was performed, but the ankylosis recurred. The author thinks that while arthroplasty may be brilliantly successful in joints ankylosed by purely traumatic lesions, it is liable to fail in joints ankylosed as a result of bacterial infection. J. Allan.

The treatment of deformities of the spine caused by poliomyelitis (*'Journ. Amer. Med. Assoc.,'* 1917, LXIX, *p.* 787).—**R. A. Hibbs** reports 8 cases in which a fusion operation was performed with good results. The operation consists in elevating the periosteum of the vertebræ from the adjacent bases of the spinous processes and the adjacent edge of the laminæ to the ventral side. A small piece of bone is elevated from the laminæ and turned down, its free end resting on the one next below it. The spinous processes are then partially divided with forceps for that purpose, and broken down so that the tip of one comes in contact with the base next below it. This completes all that is necessary to do to the bone. The periosteum and ligament which have been separated are then brought together and sutured in the centre with catgut. Immediately after operation a brace or jacket is applied, maintaining as much correction as is comfortable to the patient. The patient is kept in bed for eight weeks, and the brace is worn for one year. In six of these cases there was conspicuous deformity, and such weakness in three that the patients could not sit up unaided, much less walk. All of them have been given a much better posture, much more stability, and, it is believed, protection from a progressive increase of deformity. T. R. Whipham.

The importance of the ligaments of the ankle in correction of congenital club-foot (*'Journ. Amer. Med. Assoc.,'* 1917, LXIX, *p.* 1057).—**I. Zadek** and **E. L. Barnett** draw attention to the necessity for dividing

the posterior ligament of the ankle-joint in addition to the tendo Achillis in cases of congenital talipes equinus. By this means the foot can be pushed into dorsal flexion and the os calcis tilts up to a normal degree.

T. R. Whipham.

Ophthalmology.

Familial head nystagmus in four generations associated with ocular nystagmus (*'Journ. Amer. Med. Assoc.,'* 1917, lxix, *p.* 773).—**N. S. Yawger** gives an account of a family of Russian Jews in which combined nystagmus of the head and eyes occurred in at least four generations and persisted throughout life. The first known cases in the family were two brothers, one of whom died at the age of 86, and had eight children. Most of them died young, but one daughter who had nystagmus lived to be 80, and another who did not have the disorder transmitted it to two sons by different husbands; her daughter is unaffected. The elder son is neuropathic and stammered badly as a child. He has eight children, four of whom have been stammerers, but none shows the combined nystagmus, although in one daughter extreme excursion of the eyes causes a horizontal nystagmus. The younger son is somewhat dwarfish, and has two sons and a daughter. The older son is normal, but the other children are twin and undersized, and the girl has the familial affection.

T. R. Whipham.

The ætiology of congenital ophthalmoplegia (*'New York Med. Journ.,'* 1918, cvii, *p.* 992).—**Editorial.**—This affection is twice as common among boys as girls, and is hereditary and familial in half the cases. It is always congenital, and there is usually no history of difficult labour. Occasionally other malformations are present, as closed fontanelles, etc. The bodily development is sometimes retarded. A family history of syphilis, tubercle and alcoholism seem to have no influence. Data for establishing the ætiology are wanting at present. J. Porter Parkinson.

Energy metabolism of an amaurotic family idiot (*'Amer. Journ. Dis. Child.,'* 1918, xvi, *p.* 39).—**F. B. Talbot** investigated the energy metabolism of an amaurotic family idiot girl, aged 2 years and 4 months. She was extremely thin and emaciated, and moved in bed only when disturbed by sound or touch. The amount of muscular activity during the day was so slight as to be almost negligible, except during convulsions. The metabolism was found to be very low when compared to that of normal infants of the same weight or same age. J. D. Rolleston.

Brittle bones and blue sclerotics (*'Gazz. d. Osp.,'* 1918, xxxix, *p.* 367).—**A. Marconi** refers to the cases reported by Rolleston (*vide* British Journal of Children's Diseases,' 1911, viii, *p.* 202), Adair-Deighton (*ibid.*, 1912, ix, *p.* 376), Peters (*ibid.*, 1913, x, *p.* 522), Hoffmann (*ibid.*, 1916, xiii, *p.* 317), and others, and records the following personal case. A soldier, aged 21 years, was admitted to hospital for a fracture of the left external malleolus which had occurred without appreciable cause during marching. There was a history of several previous fractures—first at the age of 3 years in the lower third of the right tibia, and a few months later at a corresponding point of the left tibia, on both occasions as the result of a fall. At the age of 4 years he fell off a chair and fractured the right

arm a little above the elbow, and shortly afterwards the last phalanx of the middle finger of the left hand. Apart from the fractures the X rays showed nothing abnormal in the bones. The patient also presented a deep turquoise coloration of the sclerotics. None of the family were similarly affected. J. D. Rolleston.

Blue sclerotics, brittle bones, and deafness (*'Arch. f. Ophth.,'* 1918, xcv, *p.* 81).—**J. van der Hoeve** and **A. de Kleyn.**—In a family consisting of 22 persons, 9 presented the triad of blue sclerotics, brittle bones, and otosclerosis, 1 had otosclerosis and brittle bones only, and 1 otosclerosis and blue sclerotics only. In addition to fractures the patients also showed deformities of the bones, *e.g.* curved tibiæ, kyphosis, lordosis, and scoliosis. Two had had dislocations. Almost all were small and delicately built. Most had errors of refraction. Embryontoxon was not observed. In 7 of the abnormal members of the family the second and third toes of both feet had grown together, and one of them had also syndactyly of the third and fourth toes. In another family of 6 persons examined by van der Hoeve 3 presented the complete triad, 1 had blue sclerotics only, and 2 had blue sclerotics and brittle bones without otosclerosis (*cf.* British Journal of Children's Diseases, 1911, viii, *p.* 202; 1912, ix, *p.* 376; 1913, x, *p.* 522; 1914, xi, *p.* 307; 1916, xiii, *pp.* 25 and 317; and 1917, xiv, *p.* 298). J. D. Rolleston.

Blue sclerotics with brittle bones (*'Albany Med. Annals,'* 1918, xxxix, *p.* 169).—**J. B. Harvie** reports a case in a boy, aged 8 years. Father is a well-developed man and in good health. Mother had blue sclerotics and fractured her humerus in early life; this united firmly with no resulting disability. She died from carcinoma of the uterus when forty years of age. She had no miscarriages; there are five children—four boys and one girl. The second eldest boy has blue sclerotics, and has had multiple fractures. The other members of the family are healthy and apparently perfectly normal. In the present case the boy is somewhat undersized, and is rather poorly developed. All the fractures have occurred on the left side—the left tibia and fibula, the forearm, the end of the humerus, the femur and the clavicle. Some have been associated with a known injury and some have not. The bones in the vicinity of the fractures are exceedingly porous and seem to possess little mineral deposit. They are more transparent than usual, as shown by the X rays. They are soft, bend easily, and assume a sickle shape. The cortical substance is lacking and the bones are exceedingly slender, particularly the clavicles and fibulæ. The latter are about the size of an ordinary knitting-needle. The muscles and the ligaments show lack of tonicity. The joints have a wide range of motion, so that the legs may be thrown in all directions. There is marked hypotonia. The sclerotics present a pale china-blue appearance. A brief review of some previously published papers is appended. J. Allan.

The ætiology of phlyctenular conjunctivitis (*'Amer. Journ. Dis. Child.,'* 1918, xv, *p.* 81).—**W. S. Gibson.**—Clinical observations on 92 cases of phlyctenular conjunctivitis revealed the presence of tuberculosis in 90 cases as shown by a positive von Pirquet's test. In the two giving a negative reaction the diagnosis was doubtful. No other condition, either local or general, could be found with sufficient frequency even to suggest any direct influence in producing the disease. Phlyctenules were produced

experimentally in tuberculous rabbits in 8 cases, 6 times as a complication of Calmette's reaction, and twice without any local irritation. Numerous attempts to produce the condition in non-tuberculous animals failed. The microscopical findings suggested a tuberculous origin. Gibson concludes that all clinical, experimental and pathological evidence points to tuberculosis and tuberculosis alone as the cause of phlyctenular conjunctivitis.

J. D. Rolleston.

Membranous conjunctivitis following a squint operation (*'Lancet,'* 1917, II, *p.* 348).—**S. J. Taylor** performed an advancement-cum-tenotomy operation for a convergent squint in a girl, aged 11 years. Two days later the upper eyelid was a little more swollen and glazed than is usual, and the advancement-wound region did not look quite nice. This was soon succeeded by a definite, dirty, membranous area, which was diagnosed clinically as diphtheritic, though no bacteriological examination was made. Antitoxin was injected hypodermically, quinine lotion was used freely and frequently, and the condition slowly cleared up. The general systemic disturbance was very slight. The patient had had diphtheria last Christmas, but this attack apparently did not confer immunity against the local affection. The child's home environment is most unsatisfactory, the ground behind the house being soaked with sewage, and there being no drainage system there.

J. Allan.

A case of injury to the eye by the bursting of a liquid-core golf-ball (*'Med. Journ. Austral,'* 1918, I, *p.* 302).—**J. C. Halliday**.—A boy, aged 10 years, was sawing through a golf-ball when some white fluid from its interior flew up into his right eye. Marked swelling of the lids and chemosis of the conjunctiva together with photophobia and a serous discharge resulted; a fortnight afterwards the cornea became steamy. After about five weeks the eye was becoming normal. (*Cf.* British Journal of Children's Diseases, 1916, XIII, *p.* 319.—Ed.). F. R. B. Atkinson.

Primary unilateral optic atrophy in a child following fracture of the skull (*'Journ. Amer. Med. Assoc.,'* 1918, LXX, *p.* 1595).—**A. Bray.**—A girl, aged 4 years, was knocked down by a motor truck and rendered unconscious. X rays showed a fracture of the base of the skull and of the frontal bone into the frontal sinus. No ophthalmoscopic examination was made until five months after the fracture, when the right disc was found to be well defined, somewhat excavated and blueish pale, and the retina normal. Primary optic atrophy is very rare in children, and probably never occurs as a constitutional condition as in tabes, multiple sclerosis, and cerebro-spinal syphilis in adults, but may be caused by trauma. The trauma may affect the nerve directly, causing laceration, or indirectly as the result of some pressure resulting from the injury. The fracture may also involve the orbital bones. The prognosis is very grave, as blindness usually ensues.

J. D. Rolleston.

The cause and prevention of myopia (*'Lancet,'* 1918, I, *p.* 137).—**F. W. Edridge-Green** maintains that the primary and essential cause of myopia is an obstruction of the outflow of the fluid of the posterior lymph passages, which, increasing the intraocular tension, distends the sclerotic and causes myopia by elongation of the eyeball. The posterior portion of the sclerotic is a weak spot, which will give way on undue pressure. This

is particularly likely to occur whilst the eye is soft and growing, but under great stress appears to occur at almost any age. Any cause which will increase the intraocular tension by obstructing the outflow should be avoided in any case of commencing myopia so as to stop increase in the myopia and in advanced cases detachment of the retina. Careful regulation of exercise, especially in the debilitated, is necessary. Exercises which, whilst very easy for some, are accomplished by the patient with the greatest difficulty and strain, should be forbidden. Exercises which after inquiry are found to cause a feeling of pain and tension in the eyes should be strictly forbidden, but it is not necessary to keep the patient from books. It should be remembered that a myopic eye is a damaged eye, and should not be unduly strained. This is particularly the case when the myopia has just occurred or is still increasing. The same precaution should be taken in case of illness, like measles, etc. Patients should not be kept longer than possible in a recumbent position, and exercises which are likely to increase the intraocular tension should be forbidden until they have quite recovered.

J. Allan.

Review.

Transactions of the American Pediatric Society. Twenty-ninth Session, May, 1917. Edited by Oscar M. Schloss, M.D. Vol. xxix. Pp. 255.

This well-printed and illustrated volume contains thirty-three papers, some published already, on very various subjects which were read and discussed at the annual meeting at the Greenbriar, White Sulphur Springs, W.V.A. Among the longer papers Dr. Fritz B. Talbot's account of the twenty-four-hour metabolism of two normal infants with special reference to the total energy requirements embodies the results of a research begun five years before with Benedict. Dr. Abt contributes a valuable article for future reference on congenital skin defects which are most often seen on the scalp; he reports a case and collects thirty-five cases from other sources with a number of illustrations. An experimental investigation on the focal lesions produced in the rabbit by colon bacilli isolated from cases of pyelocystitis by Helmholz and Beeler shows that the intravenous injection of these bacilli sets up typical pyelocystitis in rabbits. There are three papers on acute poliomyelitis and others, such as Herrman's on three cases of sporadic cretinism in one family, that are of special interest and value. H. D. R.

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